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FATTY DEGENERATION OF THE CORNEA

(NEUTRAL AND LIPOID)

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In the ARCHIVES for May 1936 I [1] discussed two cases of corneal degeneration. One was definitely an instance of fatty degeneration, as proved by the demonstration of predominant neutral fat in histopathologic preparations. The other (case 1 in the article referred to) was regarded as a possible example of calcareous degeneration by reason of certain associated features, e. g., recurring inflammatory foci and hypercalcemia. This surmise has proved incorrect, and as I have since had the opportunity of investigating the change in the tissues in this case it is of interest to record the result.

The patient, Mr. J., on leaving the Government Ophthalmic Hospital on Feb. 13, 1935, returned to Malaya. Vision in each eye was within the normal limits. Owing to failure of postal communication, my report as to the nature of the condition and my suggestion that our respective records should be amalgamated and published did not reach Dr. Viswalingam, who had sent the patient to me. Consequently notes were published separately, mine, as has already been cited, and Dr. Viswalingam's.[2] In the latter article the patient's history was briefly sketched, and it was noted that after his return to Kuala Lumpur from Madras the left eye had to be removed, as it was blind and painful. The specimen was sent to London for pathologic investigation, and the report is quoted in full. The diagnosis made from the appearance was "sclerosing keratitis profunda." On February 13 I had suggested keratoplasty as the most hopeful line of treatment when both eyes became blind, and so, when the remaining eye lost vision, owing to the extension of the yellow infiltration throughout the whole cornea, Dr.

To be read in continuation of "Degeneration of the Cornea, Calcareous (?) and Fatty," published in the May 1936 issue of the ARCHIVES, page 803.

1. Wright, R. E.: Degeneration of the Cornea, Calcareous (?) and Fatty, Arch. Ophth. **15**:803 (May) 1936.

2. Viswalingam, A.: A Case of Sclerosing Keratitis Profunda, Brit. J. Ophth. **20**:449 (Aug.) 1936.

Viswalingam sent the patient back to me for corneal grafting. The patient arrived in Madras on July 18, 1936.

FURTHER NOTES ON CASE

Ophthalmic Examination.—The right cornea was completely infiltrated by yellow material with the exception of a small semi-opaque area near the limbus in the superior nasal quadrant. The corneal epithelium was smooth. The eyeball at this time looked very congested, but the patient said that only occasionally did he have these attacks of redness. He was taking large doses of sodium salicylate (30 grains [2 Gm.] four times daily) and considered that it controlled them. He still had recurrent inflammatory foci, which were present in the epitrochlear region, the metatarsophalangeal joint of the great toe and near the upper part of the tendo Achillis on each side, but they were not so definite as formerly. While the patient was under observation for a few days the sclera showed changes varying from pallor to intense redness. When in the pallid state it was observed to be definitely thinned out in the region of the ora serrata. The patient was reported to have had slight rises of tension during the congestive attacks in the past year. Apparently there was a difference in tension between the congested state and the more pallid state, but the rise was not to an abnormal extent, since, even in congestion the tension as measured with the Bailliart tonometer was below the average normal. The reason for these congestive attacks could not be determined. The patient had used pilocarpine for their relief, but it is doubtful whether this drug influenced the condition.

On examination with the corneal microscope it was perfectly clear that there were iridescent plates of some sort (not needles) in the middle layers of the substantia propria.

Examination of the Blood.—The blood calcium content was somewhat high, since the patient had given up the calcium-free diet which I had formerly advised. The blood cholesterol content was high, the figure being 294.2 mg. per hundred cubic centimeters of plasma. The fatty acid content of the blood was 370.8 mg. per hundred cubic centimeters of plasma; the serum calcium content was 13.6 mg. per hundred cubic centimeters, and the serum phosphate content 2.84 mg. A complete roentgen examination of the skeleton showed no change in the bones since the patient's visit of more than a year previously.

Captain T. W. Barnard, director of the Barnard Institute of Radiology, Madras, and Dr. M. J. S. Pillai, the medical superintendent, made these examinations.

Operation.—A keratoplasty was undertaken in the hope of giving the patient some vision, since this had dropped to perception of movements of the hand close to the face.

On July 25 a graft 7 mm. in diameter was taken from a donor of the same blood group and placed in an aperture 7 mm. in diameter made with a trephine in the center of the patient's cornea. It was seen on removing the disk from the patient that the cornea was at least one and one-half times the usual thickness. The chamber was shallow. There was an incipient cataractous condition of the lens; the iris looked normal, and the pupil was contracted. The graft was retained in place by three double waxed threads placed from limbus to limbus at angles of 60 degrees to each other, making six spokes radiating from the center of the graft, in accordance with my usual technic. The graft took well in spite of the discrepancy in thickness; the pupil was kept contracted with pilocarpine. There was some delay in the formation of the chamber, and anterior synechiae with the junction of the graft formed in several positions. The stitches were removed on the fifth day after the operation, and on the tenth day the graft was clear and

the patient had vision of 2/60. It was considered advisable to free the synechiae. This was done on August 13. The operation proved to be a mistake, as some blood clot formed in the pupil and diminished the vision. The freeing of the synechiae also caused a small detachment of Descemet's membrane, which started a haziness in the graft, with some vascularization. It looked as if this might determine permanent opacification of the graft, so that the prognosis for sight was bad. (Extraction of cataract would have been required, of course, in a short time had the graft kept clear.) In view of the recurring attacks of congestion

Fig. 1.—Photomicrograph of a section of the cornea of Mr. J., showing lipoid change associated with the deposition of plates of cholesterol.

and the thinning out of the sclera, it seemed highly probable that a staphylomatous condition of the sclera would eventually take place. The cornea was bad soil on which to plant a graft, although in the first instance there was a promise of some success. On August 31, when the patient was allowed to go home, his vision had definitely improved, and he could tell whether the hand was open or closed at 3 meters. He could count fingers at 50 cm. He was depressed at the result of the freeing of the synechiae but pleased to see at all.

Histopathologic Examination.—The disk of the cornea was immediately subjected to histopathologic investigation by Dr. T. Bhaskara Menon, pathologist of

the Stanley Medical School, Madras. His report confirmed the slit lamp observations at the time of this second visit, namely, that there were crystalline plates in the substantia propria. At the time of the patient's first visit the appearance was more suggestive of needle-like crystals than of plates, but as cholesterol may occur as either needles or plates, possibly the form of the deposit may have altered. The report, which follows, speaks for itself.

"I have examined the specimen of the cornea both by frozen sections and after paraffin embedding. Histologically the slides show that the surface epithelium is almost normal. Bowman's membrane is unaffected, except that it is separated in places from the epithelium. The substantia propria just beneath shows irregular oval slits which become more marked deeper down. These are spaces left by some crystalline substance which has been dissolved out. Round these slits the fibers of connective tissue are swollen and slightly hyaline, with compressed nuclei. Here and there round and oval vacuoles can be made out in the fibers. Deeper down, cellular infiltration becomes marked; the cells are mononuclear and eosinophile, with a few polymorphonuclears, all having a perivascular distribution; here the slits left by the crystalline deposit are more irregular. This cellular infiltration is most marked in the deeper layers. Frozen sections, stained and unstained, show that the crystalline deposits are surrounded by fine globules of neutral fat; these take the sudan III stain; the crystals do not take the stain. When teased out on a slide and examined they appear as characteristic rectangular plates with broken edges typical of cholesterol. They give the color reaction with acetic anhydride and sulfuric acid. I cannot find any trace of calcium deposit by von Kossa's stain. With osmic acid, black globules of fat can also be demonstrated in close relation to the crystals. Some are collected close to Descemet's membrane."

Figure 1 shows some of the changes just described.

COMMENT

It was obvious as the result of the second investigation of this case that my former notion as to its nature was incorrect. Even though the patient had hypercalcemia, attacks associated with inflammatory foci (the eye being also affected) and, in one of the nodes, an excess of tissue calcium, the corneal deposits were certainly not calcareous. The clinical picture might at first have suggested a gouty process, but this is ruled out by various findings. The abundance of cholesterol in the cornea suggests a third group of metabolic derangements, and this is supported by the hypercholesteremia. Is it possible that the inflammatory foci were associated with the deposition of lipoid? If so, the general condition would fall into the group of lipoidoses associated with defective lipoid metabolism, e. g., xanthomatosis and the syndromes associated with the names of Schüller and Christian, Gaucher, and Niemann and Pick. The general picture need not, however, concern one at the moment; there is hardly enough evidence to discuss it usefully. The corneal condition, on the other hand, is most interesting, as it affords a good example of one variety of lipoid degeneration or infiltration, namely, that associated with the deposition of plates of cholesterol. Although to the naked eye the appearance suggested a neutral fatty change, clinically rather like that in case 2 in my article

already cited, in case 1 there were histopathologic changes markedly different from those in case 2. In case 2 there was much more evidence of neutral fat, although mixtures of fatty acids and a few crystalline needles (of doubtful composition but not calcium) were present. In case 1 there was little evidence of neutral fat, but there was definite deposition of plates of cholesterol in spaces between the lamellae of the substantia propria. The clefts where the plates have disappeared are a marked feature of the histopathologic preparations. The tissue reaction was to a considerable extent associated with the deposition of the plates and was in part a foreign body reaction somewhat similar to the tissue reaction in the xanthomas. Both cases are, however, examples of those of a group of corneal degenerations which come under the generalized title of fatty degeneration or, better, perhaps, lipin degeneration. It may be helpful to think of this degeneration in two subgroups: (*A*) neutral fatty degeneration of the cornea (in which neutral fat predominates) and (*B*) lipoid degeneration of the cornea (including deposits of cholesterol, both crystalline (plates or needles) and amorphous cholesteride (such as coarse cholesterol, fatty acid esters, and finer intracellular or intercellular deposits, e. g., those of arcus senilis).

Cases of the *A* type are rare; in fact, fatty degeneration of any part of the globe associated with a predominant neutral fat change is rare. When fatty degeneration occurs in the globe the mode is ordinarily lipoidal, just as in certain other parts of the body, e. g., the aorta. I need not digress here in order to theorize on this theme.

Cases of the *B* type are not rare; for instance, arcus senilis is an everyday sight. In case 1 the condition was evidently a clinical variety of this subgroup; in the syndrome assumed it is probably uncommon, although in the degenerations of old corneal lesions the deposition of plates of cholesterol or needles is hardly so rare as one might judge from the infrequent references in the literature. One sees lipoid degeneration also from time to time as a yellow infiltrative process in keratoconjunctivitis eczematosa, trachoma and the interstitial keratitis of the granulomatous diseases.

I have recently seen a third clinical variety of corneal degeneration which falls into this subgroup. In this instance the color of the material was white, without a suggestion of yellow. The process was deep in the substantia propria, and there were deep vascularization, slight but definite interference with sensibility, and a completely clear narrow marginal ring, with the opacification starting where one ordinarily observes the arcus senilis. In fact, the condition might have been regarded as arcus senilis which had become excessive and irregular so that the advance of the opacity toward the center was not uniform

but was more intense in some quadrants than in others (fig. 2). The central portion of the cornea was not encroached on to any great extent but was faintly hazy. The interesting feature in the case was that with the corneal microscope the advancing edge of the denser areas showed definite needle-like crystals. (One cannot be sure of the exact nature of needle-like crystals, since substances other than cholesterol and fatty acid are met with in tissues in this form.) There were no associated symptoms of any importance—no jaundice and no glycosuria were present, and the blood chemistry was normal. This case recalled Dr. Parker Heath's [3] article in the ARCHIVES. It is not quite clear to me whether he wished to indicate a distinct clinical entity of metabolic etiology, in addition to suggesting that a number of corneal lesions which are now classified as separate entities might be grouped more accurately as lipin interstitial keratitis. There are, of course,

Fig. 2.—Corneal degenerative changes in the right eye (left) and the left eye (right) associated with the deposition of needle-like crystals (cholesterol?).

many corneal lesions in which lipin or lipoid degeneration takes place. It is of doubtful value to regroup them under a name signifying a common pathologic process since this does not help one to understand their etiology any better. For example, keratitis disciformis to me not only signifies a peculiar shape and distribution of a corneal lesion but in the vast majority of the cases which I see is one expression of a definite clinical entity, namely, the disease superficial punctate keratitis, which I have ventured to rename keratoconjunctivitis diversiformis et uveitis anterior. My associates and I have observed over thirteen thousand cases of this disease in Madras since 1928. We feel fairly certain that we know most of its clinical characters and think that we know its etiology. I have never seen the keratitis disciformis of this

3. Heath, Parker: Lipin Interstitial Keratitis, Arch. Ophth. **13**:614 (April) 1935.

particular disease result in lipin degeneration. I mention this point merely to illustrate how Dr. Parker Heath and I, although thinking along the same lines as regards the probable frequency and importance of lipoid degeneration of the cornea, might differ as regards the value of classifying corneal diseases by grouping them under the degenerative process concerned (which, after all, is no better a basis of classification than shape, color or any other feature) rather than according to etiology. My inclination would be to say that lipoid degeneration is a type of degeneration not infrequently met with in certain corneal conditions of unknown etiology now regarded as separate entities. I would also feel inclined to use the term lipoid degeneration rather than lipin degeneration, for lipin degeneration embraces neutral fatty degeneration, and this is rare, in my experience.

A fourth member of this subgroup is a distinct variety of primary lipoid corneal degeneration, which I [4] originally described in the *American Journal of Ophthalmology*. This is a relatively common clinical degeneration, formerly spoken of in this clinic as "corneal dystrophy of old persons" but now recognized to be a degeneration sui generis and not necessarily of old persons. Since my original description of this condition I have had an opportunity of doing a keratoplasty on an affected eye. The disk removed with the trephine was 11 mm. in diameter and afforded an opportunity of investigating corneal sections of whole thickness. The patient had corneal degeneration of each eye, and vision of each eye was perception of movements of the hand. The keratoplasty done on the left side was not a great success, since the large graft became somewhat steamy and there was a cataractous condition of the lens. Extraction of cataract would have given the patient a good practical visual improvement, probably of the order of ability to count fingers at a few meters, but the patient left the hospital, being advised to return for broad iridectomy and extraction of cataract. Vision of the left eye on discharge was ability to count fingers at 0.5 meters. Dr. T. Bhaskara Menon again conducted the histopathologic investigation, and his report is as follows:

The sections of the cornea show that the surface epithelium is thinned out and lost in part and abnormally thick elsewhere. In the substantia propria there is an infiltration of the connective tissue cells with small rounded droplets which fuse together to form larger globules and masses as described in the *American Journal of Ophthalmology*.[4]

In the center of the ulcerated area, the fibers of the substantia propria are frayed and broken, and the deposit has assumed the appearance of irregular homogeneous masses; the droplets, as well as the masses, take a reddish color with hematoxylin. They take a blue color with nile blue, suggesting fatty acid mixtures. There is no trace of neutral fat when frozen sections are stained with sudan III or osmic

4. Wright, R. E.: Interstitial Degeneration of the Cornea, Am. J. Ophth. **19**:413 (May) 1936.

acid. The globules and irregular masses have been identified as cholesterol or/and cholesterol fatty-acid esters since they give a color reaction with acetic anhydride and sulfuric acid. I had this confirmed by Dr. A. S. Mannady Nayar, professor of biochemistry at the Madras Medical College. So the condition appears to be cholesterol steatosis, as originally suspected. As regards the site of commencement, I cannot be very definite. Descemet's membrane appears normal. In the case of Mr. J., already described—in which plates of cholesterol were asso-

Fig. 3.—Photomicrograph of a section of the cornea, showing changes due to primary lipoid or cholesteride degeneration.

ciated with marked cellular changes and capillary invasion of the cornea accompanied or succeeded the initial deposition of lipoid—we could trace the change from around the blood vessels in the substantia propria, but there is no cellular infiltration here.

Figure 3 shows some of the appearances described.

Another section shows that the epithelium outside the ulcerated area is entire and is slightly hyperplastic in places, forming papilliform buds projecting down. The deposit appears in places to be just underneath Bowman's membrane in the

substantia propria. There is distinct hyalinization of the connective tissue fibers of the substantia propria around the deposit. Slight infiltration by round cells can be made out round the vessels at one end.

This infiltration was noted near the limbus; one does not necessarily find vascularization in this type of degeneration, and the cornea surrounding a patch may be perfectly free from vessels when examined carefully with the corneal microscope.

Clinically and histopathologically this corneal malady is different from that in the two cases of subgroup B just discussed, although there is the common factor of lipoid degeneration. In the latter type of degeneration the microscopic appearance of the cornea is distinctive, examination revealing the amber-colored or oil-like masses and droplets. These are large toward the center of the patch, where the process is oldest and where the masses are big enough to push the epithelium forward in rounded elevations, here and there causing its thinning out and disappearance and leaving an eroded gap. It may be seen that between the elevations of thinned-out epithelium there are irregular areas of epithelial thickenings due to proliferation. Toward the edge of the patch the droplets become smaller until they fade away as colorless points constituting a gray haze at the periphery. This appearance is suggestive of a cloudy swelling which by analogy one might perhaps expect to precede intracellular fatty degeneration. On the other hand, it may be an intracellular edematous condition. We have as yet no definite evidence, since the available sections have not included the hazy areas. The histopathologic changes under low power magnification are such as those shown in figure 2 in my article in the *American Journal of Ophthalmology*.[4] The photomicrographs and drawing illustrating the present report, although poor, are somewhat more instructive. My recent investigation does not throw any light on the etiology of this condition, but since interstitial degeneration of the cornea—the name under which I described this condition in the *American Journal of Ophthalmology*—does not even indicate the nature of the histopathologic process, perhaps the name primary lipoid or cholesteride degeneration of the cornea might be more apt. At all events, the name will serve pro tempore to indicate a relatively common corneal degeneration seen here, and readily recognized, even in its earlier stages, by those who have observed a few cases. We have looked for correlations, without result. Apparently the lipoid metabolism may be normal, but the last patient examined, a woman (and the condition is not so common in women as in men) showed 200 mg. of blood cholesterol per hundred cubic centimeters. The sugar metabolism is not necessarily deranged.

Lipoid degeneration is, to be sure, a common postmortem appearance in this part of the world in the seats of election, viz., at the exits of the branches of the main arterial stem, but possibly not more so here than

elsewhere. The general resemblance between atheromatous degeneration of the great vessels and that form of degeneration just described suggests itself; the intimate nature of these processes is almost identical. The word atheromatous (or hyaline) has been used in referring to certain corneal degenerative conditions for a long time but not in the sense of primary degeneration. It would seem to be applied to changes taking place in old corneal lesions, and the yellow spots have been regarded as hyalin. Here let it be emphasized that the particular type of lipoid degeneration just described as cholesteride is not infrequently seen in old corneal opacities of various types. It is readily recognized by the brownish or amber-colored masses, or oil globule-like collections of cholesterol fatty-acid ester, which one sees from time to time when examining old corneal lesions with the slit lamp; but in such cases the condition is secondary to an initial corneal lesion.

One gets the impression that this is the commonest type of fatty degeneration of the cornea, whether primary or secondary, and it may be that it has been confused with hyaline degeneration. It is a matter of pathologic interest that when lipin degeneration does occur in the eyeball it most frequently assumes some variety of the lipoid mode of degeneration. One is familiar with it in the cornea, the lens, the vitreous, the retina and the optic nerve head, e. g., in the lens as cholesterin crystals and in the vitreous as crystals or the rounded bodies of Benson's asteroid hyalitis. In records of pathologic studies of diseases of the eye one seldom finds references to neutral fat except in the form of droplets associated with the predominant picture of lipoid change. That cholesterol often appears as a sequel to hemorrhage is true, but hemorrhage is not a necessary preliminary. In fact, lipoid degeneration of the cell protoplasm unassociated with hemorrhage is probably more common. It may perhaps be said of the eyeball, as of the aorta, that lipoid change is the mode of election when the concealed fat of the protoplasm of the cell is thrown out of solution by local degeneration or generalized interference with the lipin metabolism, e. g., in the lipoidoses.

That there may be types of lipoid degeneration other than those given in subgroup B goes without saying. This note is not intended to be an essay on the subject or, in fact, anything more than a record of some clinical observations with musings thereon which may interest others who have the leisure to collect data, make detailed observations and review the literature. I regret that lack of time has not permitted me to consult records bearing on the subject other than a small selection at hand.

Dr. T. Bhaskara Menon carried out the histopathologic investigations, and Colonel H. E. Shortt, of the King Institute of Preventive Medicine, Madras, prepared the photomicrographs.

HERPES ZOSTER OPHTHALMICUS COMPLICATED BY OPHTHALMOPLEGIA AND EXOPHTHALMOS

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The complications of herpes zoster ophthalmicus vary greatly among different patients. The major portion of the complications fall under four types: (1) keratitis, (2) iridocyclitis, (3) muscular palsies and (4) optic neuritis.

In about 50 per cent of all the cases the globe is affected, and in the majority of these (35 per cent of all cases) the cornea suffers.

Hutchinson stated that the globe is never affected except in cases in which the nasociliary nerve is affected, but exceptions to this rule have been reported. Thus, scleritis, superficial keratitis, keratitis disciformis, interstitial keratitis and other forms of deep keratitis are rather commonly observed. In most of these keratitides the iris is also affected, and both hypotension and hypertension have been recorded. Worster-Drought [1] stated that in 7 per cent of these cases the condition is associated with paralyses.

The third nerve is the most frequently involved, but it is unusual for the whole of the third nerve to be affected at the same time.

In a series of one hundred and fifty-eight patients Hunt [2] found twenty-four with paresis of the ocular muscles. He observed that the third nerve was affected in eighteen, the sixth nerve in five and the fourth nerve in one. Eighty of this group showed facial paralysis, and 64 per cent showed some associated paralysis in the nervous system.

Numerous cases of palsy of various nerves have been reported. Thus, palsy of the third nerve has been reported by Wallace,[3] Brissaud,[4] Zentmayer,[5] Fage,[6] Rosnoblet,[7] Gallois [8] and others; palsy of the fourth

From the Department of Ophthalmology, the Cook County Hospital and Rush Medical College, University of Chicago.

1. Worster-Drought, C.: Brit. M. J. **1**:970 (June 9) 1923.
2. Hunt, J. Ramsey: The Paralytic Complications of Herpes Zoster of the Cephalic Extremity, J. A. M. A. **53**:1456 (Oct. 30) 1909.
3. Wallace, W. T.: Ophth. Rec. **20**:119, 1911.
4. Brissaud, in Wilbrand, H., and Saenger, A.: Neurologie des Auges, Munich, J. F. Bergmann, 1921, vol. 8, p. 238.
5. Zentmayer, W.: Am. Med. **4**:1007 (Dec. 27) 1902.
6. Fage: Rec. d'opht. **31**:209, 1909.
7. Rosnoblet: Zentralbl. f. d. ges. Ophth. **15**:426, 1925-1926.
8. Gallois: Clin. opht. **14**:421, 1925.

nerve by Caspar [9] and Vogel; [10] palsy of the sixth nerve by Galezowski and Beauvois [11] and Carpenter,[12] and combinations of palsy of various nerves by numerous other authors.

Aubineau [13] reported a case of typical herpes zoster complicated by complete fixation of the globe and associated with mydriasis and no reaction of accommodation to light. The patient died of nephritis, and necropsy revealed thrombo-arteritis obliterans of the ophthalmic artery, especially of the ramifications to the gasserian ganglion. Further cases of total ophthalmoplegia were reported by Metz,[14] Velter,[15] Leplat,[16] Barrière,[17] Silcock [18] and Batignani.[19]

Batignani's case was that of a 66 year old woman with a negative Wassermann reaction in whom herpes zoster was complicated by secondary uveitis, mydriasis of the pupils and anesthesia of the cornea. In the course of two weeks this patient showed total ophthalmoplegia associated with moderate exophthalmos (16 mm. in the right eye and 19 mm. in the left). Both the ophthalmoplegia and the exophthalmos cleared up entirely in two months. Batignani attributed the total ophthalmoplegia to direct extension to the oculomotor extrinsic musculature, and the exophthalmos to atony of the extrinsic muscles, with resulting proptosis.

Exophthalmos as a complication of herpes zoster has been reported by only a few authors. G. ten Doesschate [20] recorded a case of herpes zoster ophthalmicus on the right in a man aged 52, which was complicated by marked exophthalmos and by a rise in tension to from 60 to 70 mm. of mercury. The exophthalmos was considered to be the cause of the hypertension, and a trephining was done, which lowered the tension, the exophthalmos remaining the same. The tension rose again in a few days but dropped to normal when the exophthalmos began to decrease. Ten Doesschate believed that the proptosis was the result of stimulation of the sympathetic fibers.

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9. Caspar: Arch. f. Augenh. **48**:177, 1903.

10. Vogel, F. E.: Augenmuskellähmungen bei Herpes zoster ophthalmicus, Inaug. Dissert., Leipzig, E. Lehmann, 1912.

11. Galezowski, J., and Beauvois, A.: Rec. d'opht. **28**:654, 1906.

12. Carpenter, in Wilbrand, H., and Saenger, A.: Neurologie des Auges, Munich, J. F. Bergmann, 1921, vol. 8, p. 239.

13. Aubineau: Clin. opht. **7**:513, 1914-1915.

14. Metz: Ohio State M. J. **7**:330, 1902.

15. Velter: Arch. d'opht. **43**:634, 1926.

16. Leplat: Bull. Soc. belge d'opht., no. 63, 1931, p. 39.

17. Barrière, A. V.: Arch. urug. d. med., cir. y especialid. **3**:498, 1933.

18. Silcock, in Wood, Casey: American Encyclopedia of Ophthalmology, Chicago, Cleveland Press, 1913, vol. 8.

19. Batignani, A.: Boll. d'ocul. **13**:814, 1934.

20. Ten Doesschate, G.: Paper read at the general meeting of the Netherlands Ophthalmologic Society, December 1918.

In the discussion of ten Doesschate's case, Mulock, however, reported a case of herpes zoster ophthalmicus complicated by exophthalmos which followed periodontitis. He attributed the exophthalmos to collateral edema; the tension was normal.

Vialeix, Prosper-Veil and Isnel [21] described a case of herpes zoster of the right eye of a 66 year old patient, complicated by paralysis of the third and sixth nerves and associated with exophthalmos. The exophthalmos appeared gradually following the zoster. The Wassermann reaction was positive, and the exophthalmos was thought to be due to inflammation of retrobulbar tissue.

Voisin [22] related a case of the disease in a man aged 73 who on the first day of the disease presented exophthalmos, palsy of the abducens nerve and total oculomotor paralysis. The Wassermann reaction was negative for both the blood and the spinal fluid; no definite etiologic factors were found. Mobility returned in two months.

REPORT OF A CASE

A. R., a Negro aged 40, was referred to the Cook County Hospital on June 30, 1934. He stated that three weeks previously he first noticed an eruption of blisters, which appeared over the right side of his forehead and nose. This condition became progressively worse, and three days later he first noticed a rather rapid protrusion of the right bulb. This protrusion increased in amount over the next one and a half weeks.

There was a history of chancre fifteen years previously and an "abscessed tooth" one week before the onset of the ocular trouble, but the latter infection had cleared up after three or four days, without medical aid.

Examination of the eyes showed vision of the right eye to be ability to count fingers at 2 feet (61 cm.) and vision of the left eye to be 0.8, and with the left eye the patient could read Jaeger's test type 1 at 16 inches (40.6 cm.). There was an eruption of vesicles, some of which had already dried and crusted and a few of which were secondarily infected, over the region of distribution of the first branch of the right trigeminal nerve, including the nasociliary branch, so that the lesions were found on the upper lid, on the forehead as far as the scalp and also on the nose. The eruption was sharply delimited at the midline.

The lids of the right eye were edematous, and the lower lid was ectropionized by a large roll of chemotic bulbar conjunctiva. The bulb was proptosed; the right eye showed exophthalmos of 32 mm.; the left eye, exophthalmos of 24 mm. (as tested by the Hertel exophthalmometer).

There was complete immobility of the globe. Corneal sensation was slightly diminished. There was marked ciliary injection, and the cornea was diffusely cloudy, with a clover-shaped superficial fluorescein-staining ulceration covering most of the central three fifths of the cornea. Deep infiltrates were also scattered throughout the cornea. The iris was muddy brown. The pupillary margin was adherent to the lens by dense posterior synechiae throughout almost its entire extent. The pupil measured 4.5 by 5 mm. and was irregular and fixed. The

21. Vialeix, V.; Prosper-Veil, and Isnel, R.: Ann. d'ocul. **162**:569, 1925.
22. Voisin, M. J.: Ann. d'ocul. **73**:820, 1936.

tension was normal on tactile pressure. The corneal clouding obscured all the details of the fundus. The left iris was brown; the pupil and disk were normal.

No focus of infection was found in the ears, nose or throat. The department of oral surgery reported the right lower third molar as a possible source of infection; this tooth was extracted on July 4. The remaining part of the physical examination gave essentially negative results.

The urine was normal; the Wassermann reaction was markedly positive.

Local treatment consisted of the instillation of a 1 per cent solution of atropine sulfate four times a day and treatment with atropine powder to break up the synechiae, together with the application of heat. General treatment was carried out with neoarsphenamine in doses of 0.6 Gm., given at weekly intervals. Solution of posterior pituitary did not relieve the pain, but this abated spontaneously three or four days after admission.

After two weeks the superficial infiltrates were epithelized, but otherwise the findings were the same as previously and remained so for another month.

Photograph of the patient, showing herpetic lesions.

On September 9 the exophthalmometer readings were 30 mm. for the right eye; 24 mm. for the left, and slight motion of the bulb was returning. Irradiation with the roentgen rays in several half-erythema doses was applied to the right orbital region to stimulate resolution. By November 4 almost complete range of ocular motions had returned, and the conjunctival roll had receded. Vision of the right eye was 4/200, and there was marked superficial vascularization extending for 2 mm. into the cornea from the limbus around its entire circumference. However, the deep opacities in the stroma showed little absorption. The exophthalmometer readings at this time were 26 mm. for the right eye and 24 mm. for the left. The details of the fundus were still obscured by the corneal condition.

Reexamination on Dec. 9, 1936, revealed vision of 0.3 in the right eye with glasses and vision of 1.2 in the left eye with glasses. The old scars from the herpetic eruption still remained. The ocular motility was normal. The superficial vascularization was the same as previously. Numerous diffusely scattered superficial nebular corneal opacities were present.

COMMENT

The attempt to determine the cause of the ophthalmoplegia and of the exophthalmos in these cases has elicited various explanations. As early as 1871 Wyss described numerous hemorrhages in muscle substance and ascribed the muscular paresis to thrombophlebitis. It appears that the inflammation involves not only the posterior root ganglions and the nerve fibers originating from these ganglions but also the skin, the mucous membrane and other structures supplied by these nerves.

Various authors have called attention to the increased prevalence of herpes zoster in syphilitic patients, and it is difficult to say whether the case reported in this paper was one of the epidemic type occurring in a syphilitic patient or one of symptomatic type due to syphilitic basilar meningitis. However, in this case, as in Voisin's case, the clinical appearance was that of inflammation of the retrobulbar tissue.

GLAUCOMA AT THE WILLS HOSPITAL

1926-1935

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The object of this work was to analyze all cases of glaucoma in which the condition was diagnosed as such in the clinical records of the Wills Hospital during 1926 to 1935, inclusive.

The term glaucoma is applied to a variety of conditions in which the common physical sign is pathologically increased intra-ocular pressure. Some forms of glaucoma may be considered as disease entities, but etiologically, pathologically and clinically they vary so much that they should not be considered as forms of the same disease. However, the present knowledge of the etiology is so meager that it appears better and certainly more practical to describe the various types of ocular hypertension generically by the name of glaucoma. Accordingly, no attempt was made (with the exception of cases of secondary glaucoma), to group the cases on the basis of etiology or pathology; they were simply listed under the diagnoses used for the purpose of filing in the hospital. These diagnoses were classified as follows:

a. Primary glaucoma
  (1) Infantile
  (2) Juvenile
  (3) Acute congestive (uncompensated)
  (4) Chronic congestive (uncompensated)
  (5) Noncongestive or simple (or compensated)

b. Secondary glaucoma, classified as to etiology

In the ten year period 1,876 records of patients with conditions diagnosed as glaucomatous were available, including records of patients seen both in the dispensary and in the wards. On the basis of a total of 242,533 patients admitted to the hospital, this represents an incidence of glaucoma of 0.78 per cent. Data for each patient were assembled from the records of both the dispensary and the house on an individual record card prepared for each patient.

The following items were listed in each record, an effort being made to obtain such information when possible.

1. Factors of age, sex, marriage status, occupation, race and nationality
2. History, including mode of onset, symptoms and previous treatment
3. Specific diagnoses, classified as:
    a. Primary glaucoma
        (1) Infantile
        (2) Juvenile
        (3) Acute congestive
        (4) Chronic congestive
        (5) Noncongestive or simple
    b. Secondary, classified as to etiology
4. State of refraction
5. Visual acuity, visual fields and intra-ocular tension
6. Local ocular findings, including the condition of the cornea, anterior chamber, iris and pupil, lens, nerve head and retina
7. General physical status of the patient, with reference to the presence of syphilis, arteriosclerosis, hypertension, cardiorenal disease, diabetes, arthritis, imbalance of the endocrine glands or of the sympathetic nervous system, etc.
8. Laboratory data
9. Treatment
    (*a*) Operative
    (*b*) Nonoperative
10. Subsequent course, as checked by visual acuity, fields, tension and development of complications (infection, cataract)
11. Present status, both physical and sociologic, of the patient, determined from hospital records and from follow-up study of the patients living in Philadelphia

Of the 1,876 patients, 768 were residing outside of Philadelphia. No attempt was made to contact these persons. Letters were written to 1,008 patients in Philadelphia in whose cases it was felt that further information would be desirable; 573, or 56.8 per cent, of the letters were returned by the post-office as undeliverable. The remaining 535 patients were visited in their homes by field workers to obtain further information about the status of the patients and to persuade them to visit the hospital for follow-up study. Of the latter group, 264 patients actually reported, of whom 147 were found to have true primary glaucoma.

It is pertinent here to compare the figure of 0.78 per cent for the incidence of glaucoma in a large ophthalmic hospital situated in a metropolitan area and drawing patients from a surrounding well populated territory with figures from various countries as given in the literature.

The data in table 1 were gathered from various sources and show the incidence of glaucoma in various countries of three continents.

The data in table 2, taken from Nelander's [1] report (1933), are more authentic, since the number of actual cases of ocular conditions reviewed and the number of cases of glaucoma found are given.

The general uniformity of these figures is striking and would indicate that glaucoma bears a fairly fixed ratio to the general population.

TABLE 1.—*Incidence of Glaucoma in Various Foreign Countries*

Country or City	Investigator	Year	Percentage
Asia			
Indo-China	Bargy	1929	2.00
Turkestan	Pokrowsky	1924	2.10
Siberia—Russia	Cykulenko	1930	1.40
Kasan—Siberia	Batarckukov	1929	2.50
India—Madras	R. E. Wright	1929	2.60
India—Madras	R. E. Wright	1930	2.00
India—Madras	R. E. Wright	1931	1.60
North China—Peiping	Pillat	1932	1.11
Japan	Joshida	1927	0.86
China	Pillat	1935	1.17
Africa			
Egypt	McCallan	1922	1.75
Tunis—Zarkum	Cuénod and Nataf	1928	2.00
Europe			
Denmark	Peters	....	2.40
Berlin	Graef	....	1.27
Wiesbaden	Peters	....	1.48
Vienna	Peters	....	1.26
Breslau	Peters	....	1.84
Göttingen	Schmidt	....	0.40
Tübingen	Schüssele	....	0.73
France	Peters	....	1.30
Paris	De Wecker	....	1.17
Portugal—Lisbon	da Gama Pinto	....	1.06
Greece—Athens	Bislis	....	1.25
Poland	Mikulinska	....	0.64

TABLE 2.—*Incidence of Glaucoma in Patients with Ocular Conditions (Nelander)*

Author	Number of Investigated Cases of Ocular Conditions	Cases of Glaucoma	Percentage
Ahlström	109,621	1,208	1.10
Coppez	148,000	1,511	1.02
Schmidt-Rimpler	55,146	688	1.24

SECONDARY GLAUCOMA

Cases of secondary glaucoma comprise a distinct group and should not be classed and described as cases of glaucoma, for the condition is simply intra-ocular hypertension with attendant symptoms due to some more or less definable cause. It may vary from simple hypertension closely resembling mild primary glaucoma through all gradations to a violent burst of hypertension with all the characteristics of acute con-

1. Nelander: Acta ophth. **11**:370, 1933.

gestive glaucoma. All cases of intra-ocular hypertension should be studied with the purpose of determining whether they should be grouped with cases of primary glaucoma or with those of secondary glaucoma. On the basis of the data available in the hospital records an attempt was made to do this, and 413 cases were classified as secondary—22 per cent of the total series of cases of gluacoma. This is to be compared with Carvill's [2] figure of 40 per cent of cases of secondary glaucoma in her series of cases of glaucoma observed in the Massachusetts Eye and Ear Infirmary.

TABLE 3.—*Grouping of Cases of Secondary Glaucoma According to Etiology*

Cause	Number of Cases
Trauma	101
Cataract	67
Iritis	57
Uveitis-choroiditis	46
Keratitis, corneal ulcer	30
Iridocyclitis	27
Dislocated lens	13
Intra-ocular tumor	15
Retinal vascular lesions	12
Retinal detachment	3
Congenital defect of the iris	1
Postoperative secondary glaucoma	27
Unclassified	14

Table 3 indicates a grouping according to etiology. The individual groups in this table may be analyzed as follows:

Trauma.—This was found to be a factor in producing secondary glaucoma, either with or without perforating injury to the eyeball, in 101 cases. From the standpoint of this review it is significant only to list the forms and the eventuality of treatment. Thirty-five patients received only medical treatment. Operative procedures were performed on 45; the operations included the following:

Iridectomy	12
Paracentesis	4
Elliot trephine operation	5
Modified Lagrange operation	2
Enucleation	22

Two patients were found to have syphilis, which may or may not have been a factor. The end-results of treatment in this series are shown by the fact there was loss of vision of the affected eye in 35 per cent of the cases.

Cataract.—Swelling of the lens during the course of senile cataract or following the development of traumatic cataract caused secondary glaucoma in 67 cases. In 25 of these the condition was traumatic in

2. Carvill, M.: Tr. Am. Ophth. Soc. **30**:71, 1932.

origin; in 3 it occurred in association with complicated cataract, in 2 in association with diabetic cataract and in the remainder in association with cataract of the senile type. In 5 cases an Elliot trephine operation was necessary to reduce the tension, in addition to extraction of the lens. In this series enucleation was performed in 14 cases—a figure showing poor results in 21 per cent of the cases.

Iritis.—Glaucoma that developed during the course of disease of the iris was present in 57 cases; in 16 of these the patient had a positive Wassermann reaction and the condition was recorded as syphilitic in origin. Blindness resulted in 16 eyes—an incidence of 28 per cent.

Iridocyclitis.—The only causes determined for this condition were syphilis (1 case) and tuberculosis (1 case). Poor visual results occurred in 13 cases, or 49 per cent.

Choroiditis.—There were 46 cases in which choroiditis was a factor in causing secondary glaucoma. In 11 of these the condition was syphilitic and in 2 tuberculous. Loss of vision occurred in 6 eyes, or 13 per cent.

Disease of the Cornea (Keratitis, Corneal Ulcer).—In 30 cases secondary glaucoma was due to disease of the cornea; syphilis was a factor in 6. Loss of vision occurred in 6 eyes, or 20 per cent.

Dislocated Lens.—In 14 cases dislocation of the lens was a factor. In 10 of these cases the dislocation was traumatic. There was loss of vision in 9 cases, or 64 per cent.

Intra-Ocular Tumor.—Fifteen cases of secondary glaucoma due to intra-ocular tumor were listed, in 4 of which the growth was melanotic sarcoma of the choroid, in 1 spindle cell sarcoma and in 3 mixed cell sarcoma. Fourteen eyes were enucleated, 1 patient refusing operation.

Retinal Vascular Disease (12 cases).—Only 2 instances of secondary glaucoma following thrombosis of the central retinal vein were found—a surprisingly low incidence. There were 4 cases of intra-ocular hemorrhage secondary to vascular disease and 1 case of glaucoma following obstruction of the central retinal artery. In the remaining cases secondary glaucoma was associated with arteriosclerotic and hypertensive retinitis. Two patients were syphilitic.

Postoperative Secondary Glaucoma (27 cases).—In this group 12 patients had been previously operated on at other institutions and were seen in the Wills Hospital with the condition established. In 15 cases the condition was noted after operation in our own institution. The primary operative procedures and the number of cases were as follows:

Iridectomy	3
Capsulotomy	1
Discission	1
Extraction of cataract	22

This compares with a figure of 24 cases reported by Fox [3] (1936) from the Bellevue Hospital of New York for the period 1923-1934. In our series of 27 cases there was visual failure in 11—an incidence of 40 per cent.

Other Factors.—Retinal detachment was found associated with secondary glaucoma in 3 cases, with no apparent associated intra-ocular tumor. One case of secondary glaucoma in a patient with aniridia was observed. There were also 14 cases in which no diagnosis as to the etiology of the secondary rise in tension was available.

Comment.—On reviewing the series of cases of secondary glaucoma as a whole, it is found that trauma as a factor was present in 136 cases, an incidence of 32.9 per cent; syphilis was present in 31.5 per cent. The seriousness of the development of secondary glaucoma is made obvious by the figures for loss of vision, which range as high as 64 per cent in the cases of dislocated lens. If the cases of intra-ocular malignant growth requiring enucleation are omitted, the incidence of loss of vision in the series of cases of secondary glaucoma is 34 per cent.

It is our impression from reviewing these cases and also from a study of the records of the patients whose condition was diagnosed as primary glaucoma that in a goodly percentage of the latter the condition would be found actually to have its origin in some antecedent ocular disease. It would seem that the figure of 22 per cent for the incidence of secondary glaucoma is a minimum one, which could be increased by closer scrutiny of cases of glaucoma with a view to exposing cases of low grade, subthreshold uveitis acting to produce a secondary rise in tension.

CONGENITAL GLAUCOMA (BUPHTHALMOS)

Under this heading are classified cases of glaucoma due to prenatal defects in the development of the eye in which the filtrating mechanism is affected. Buphthalmos may be present at birth and progress rapidly, so that the condition is apparent and the patient is brought to the attention of the ophthalmologist during the early months of life or, in some instances, the anomaly may be of lesser degree and the symptoms of increased intra-ocular pressure may develop gradually. In the latter instances, patients are first brought to the clinic at varying ages. Occasionally this condition is overlooked in the early stage and is recognized so late that it is classified as juvenile glaucoma.

The consensus in the literature is that in the majority of these cases the condition is bilateral, that males are much more frequently affected

3. Fox, S. A.: Postoperative Glaucoma, Arch. Ophth. **16**:585 (Oct.) 1936.

than females, that heredity is a factor in the development of the disease and that other congenital anomalies of the eye may be present.

Buphthalmos is a comparatively infrequent disease. Gros [4] reported 8 cases, or 0.07 per cent, among 12,000 cases observed at the ophthalmic clinic of the Hôtel-Dieu, Paris. He collected altogether 45 instances. He found that the anomaly was noticed at or immediately after birth in 60 per cent of the cases, within the first year in 13 per cent, from the first to the third year in 17 per cent and later in 9 per cent. He found both eyes affected in 63.7 per cent. Of his cases, 61.2 per cent were found in boys and 38.8 per cent in girls.

Carvill [2] reported 4 cases of congenital glaucoma in 31,648 patients with ocular conditions examined during two years—an incidence of 0.01 per cent. All of the 4 patients were white boys. The condition was unilateral and was noticed shortly after birth. One patient presented a coloboma of the iris as an associated anomaly.

TABLE 4.—*Age at Which Patients with Congenital Glaucoma Were First Seen in the Clinic*

Age, Years	Number of Patients
Under 1	8
2 to 5	6
5 to 10	4
10 to 20	6
20 to 30	2
30 to 40	2

In our series 28 patients with congenital glaucoma were discovered—an incidence of 0.011 per cent. Of these, 20, or 71 per cent, were males, and 8, or 29 per cent, were females—which strongly bears out the opinion that the condition is sex-linked and is predominant in males. All the patients were of the white race. This is somewhat at variance with Zentmayer's [5] opinion that buphthalmos is more frequent in the Negro. Table 4 denotes the age at which the patients were first seen in the clinic. There were no definite data as to the influence of heredity in these cases.

In 3 of the patients, one eye alone was affected; in the remaining 25 the condition was bilateral.

The refractive state of the involved eyes could be determined in only 3 cases and in all these it was of a high myopic nature. This finding corresponds with the general opinion that myopic defects are relatively common in buphthalmos.

No positive Wassermann reaction was found in any of the cases, so the influence of congenital syphilis apparently did not play a part.

4. Gros: Etude sur l'hydrophtalmie ou glaucome infantile, Thèse de Paris, no. 237, 1897.

5. Zentmayer, W.: Ophth. Rec. **22**:25, 1913.

The physical characteristics of the buphthalmic eyes were in most cases typical of the condition, i. e., a large cornea, a deep anterior chamber, atrophy of the iris and excavation of the optic disk. In 2 instances the lens was dislocated, and in 1 case it was calcified; 1 patient had detachment of the retina. Vision was uniformly poor in most of the cases, ranging below 3/60.

Treatment.—As the patients included in this survey were taken from the services of nine surgeons, there were a variety of treatment and a lack of uniformity that make evaluation difficult. Eight patients received treatment with miotics; 6 were subjected to posterior sclerotomy at one time or another; in 3 cases an Elliot trephine operation was performed, and 4 eyes were enucleated.

The results for only 10 patients could be secured on follow-up study. Among these vision was maintained in only 3, and in 7 the visual result was poor. This is in keeping with the general opinion that the percentage of cases in which the treatment of congenital glaucoma with miotics is successful is low and that the prognosis is generally poor.

JUVENILE GLAUCOMA

The exact definition of juvenile glaucoma is difficult, and many authorities are at variance as to the prevalence of the condition as a pathologic or clinical entity. Again, writers use different standards as a criterion for making the diagnosis. Some consider all primary glaucoma occurring in persons under the age of 35 as juvenile glaucoma; some take 40 years as the upper limit, and others 20. Glaucoma which occurs as a result of congenital defects should be excluded from this definition. It would appear that juvenile glaucoma should be considered as a disease of younger patients in whose eyes changes have developed which predispose to or excite an attack of hypertension.

Carvill [2] included in her series of cases of juvenile glaucoma those cases of primary glaucoma occurring in persons between 5 and 40 years of age, excluding cases of buphthalmos. We have adopted this grouping and have attempted to classify the records similarly.

Accordingly, 20 cases of juvenile glaucoma are reported, in 12 of which the patients were females and in 8 males. This is at variance with Löhlein's [6] report that juvenile glaucoma occurs more frequently in males, although Haag [7] stated that the predominance of the disease in one sex was not well marked in his material.

Our patients ranged in age from 18 to 32 years; none of them had had any previous treatment. No hereditary influence could be demonstrated in any of these cases, although most authors have stated that

6. Löhlein, W.: Ber. ü. d. Versamml. d. ophth. Gesellsch. **12**:97, 1913.
7. Haag, C.: Klin. Monatsbl. f. Augenh. **19**:133, 1915.

heredity shows a more strongly marked influence in juvenile glaucoma than it does in the senile form of the disease.

The refractive state was determined for only 6 of these patients, 4 being myopes and 2 hyperopes. This may have some significance, as many authorities agree that myopia is found more often in persons with juvenile glaucoma than in those with senile glaucoma. In Haag's [7] series, 31 per cent of the patients were myopes, and 28 per cent were hyperopes. In Löhlein's [6] series, 50 per cent of the patients were myopic. In Carvill's [2] cases the condition was equally distributed between hyperopes and myopes.

Most of our patients were first seen when the condition was in an advanced stage, the vision being uniformly below 6/60. An associated anomaly was found in 1 case—bilateral coloboma of the iris. Löhlein found that 50 per cent of his patients had a congenital anomaly, as did Haag.

Three patients received treatment with miotics; basal iridectomy was performed on 5, the Elliot trephine operation on 3 and the Lagrange operation on 1; 1 eye was enucleated; the remaining patients were seen only once and did not return for treatment.

The results of therapy were unfavorable in most cases.

PRIMARY GLAUCOMA

The rest of the cases are considered cases of primary glaucoma; with the information available, an attempt was made to classify the forms of primary glaucoma in detail as: (1) congestive, inflammatory or noncompensated, or (2) simple, noncongestive or compensated. Type 1 was subdivided into: (*a*) the acute congestive form and (*b*) the chronic congestive form. The groups were assembled from the data noted in the records and the original diagnoses used for the purpose of filing, although changes were made in cases in which it was felt that the subsequent developments altered the diagnosis from the simple to the congestive type.

Under the heading of simple glaucoma we included those cases in which the patient complained of only visual loss without marked discomfort, with the development of increased intra-ocular pressure, typical changes in the field and excavation of the disk to any degree. Those cases in which there were definite symptoms of headache and ocular pain and local findings of ciliary stasis, corneal edema, a shallow anterior chamber, etc., were included in the group of cases of congestive glaucoma. These cases were divided into those of acute glaucoma and those of chronic glaucoma, depending on the degree and duration of the symptoms.

One thousand, four hundred and fifteen cases of primary glaucoma, including those of all types, were noted in this study, of which 392,

or 27.7 per cent, were cases of the congestive type, and 1,023, or 72.3 per cent, cases of the simple noncongestive type. Of the group of cases of congestive glaucoma, 161, or 42.9 per cent, were observed when the condition was in the acute stage, and in 231, or 58.1 per cent,

TABLE 5.—*Distribution of Cases of Primary Glaucoma According to Type, Age Group and Sex*

Age Group	Acute Congestive (11.4%)			Chronic Congestive (16.3%)			Simple (72.3%)			Total No. of Cases (Male 48.5%) (Female 51.5%)		
	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total
40-49	7	10	17	13	15	28	58	67	125	78	92	170
50-59	37	20	57	28	37	65	120	122	242	185	179	364
60-69	26	28	54	35	53	88	188	195	383	249	276	525
70-79	9	21	30	18	25	43	124	118	242	151	164	315
80-89	1	2	3	4	3	7	19	12	31	24	17	51
	80	81	161	98	133	231	509	514	1,023	687	728	1,415

TABLE 6.—*Distribution of Cases of Primary Glaucoma According to Type, Age Group and Sex*

Age Group	Congestive (27.7%)			Simple (72.3%)		
	Male	Female	Total No.	Male	Female	Total No.
40-49	20	25	45	58	67	125
50-59	65	57	122	120	122	242
60-69	61	81	142	188	195	383
70-79	27	46	73	124	118	242
80-89	5	5	10	19	12	31
Total	178	214	392	509	514	1,023
Percentage	45.4	54.6		49.7	50.3	

TABLE 7.—*Summary of Distribution of Cases of Primary Glaucoma According to Type and Age Group*

Age Group	Acute Congestive		Chronic Congestive		All Cases of Congestive Type		Simple	
	No.	Percentage	No.	Percentage	No.	Percentage	No.	Percentage
40-49	17	10.5	28	12.1	45	11.5	125	12.2
50-59	57	35.4	65	28.1	122	31.1	242	23.6
60-69	54	33.5	88	38.0	142	36.2	383	37.4
70-79	30	18.6	43	18.6	73	18.7	242	23.6
80-89	3	1.9	7	3.2	10	2.5	31	3.2
	161		231		392		1,023	

the condition was considered to be in the chronic stage. Among the entire series of cases of glaucoma, those of acute congestive glaucoma comprised 11.4 per cent, those of chronic congestive glaucoma 16.3 per cent and those of chronic simple glaucoma 72.3 per cent. These figures, together with those showing the distribution of the cases according to age group and sex, are shown in tables 5, 6 and 7.

These figures may be compared with those found in similar studies reported in the literature. Carvill's [2] series included 52 per cent cases of noncongestive glaucoma and 46 per cent cases of congestive glau-

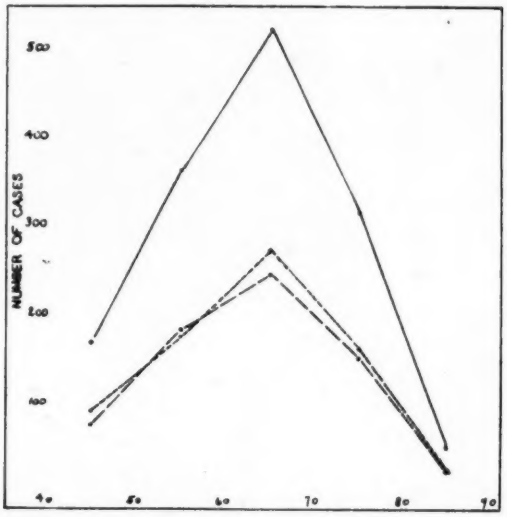

Chart 1.—Frequency of primary glaucoma at different periods of life. The long dash line indicates males; the short dash line, females, and the unbroken line, the total number.

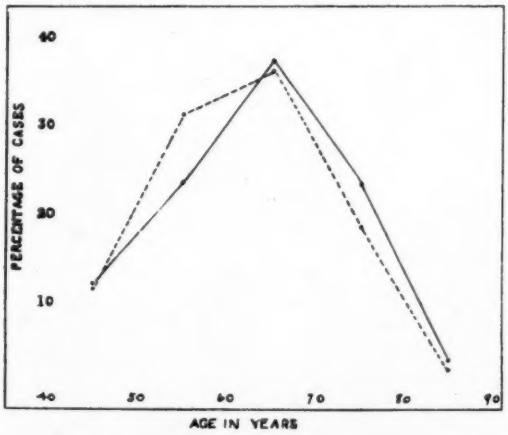

Chart 2.—Frequency of congestive and noncongestive glaucoma at different periods of life. The dash line indicates congestive glaucoma; the continuous line, noncongestive glaucoma.

coma—a greater incidence of the congestive type as compared with the incidence in our series. Weinstein,[8] in giving the results of a study

8. Weinstein, P.: Monatschr. f. Augenh. **12**:794, 1934.

of 3,214 cases observed in thirty years at the University Hospital in Budapest, reported a vast predominance of cases of the congestive type, only 306 cases of simple glaucoma having been noted. Pillat,[9] in his series from China, made no distinction except to segregate the cases of acute glaucoma as an isolated group comprising 5 per cent of his series. Nelander,[1] in Uppsala, Sweden, in a series of 75 cases, found 6 instances of acute glaucoma, 7 of chronic congestive glaucoma and 62 of simple glaucoma. Trantas,[10] in discussing a series of 428 cases studied in Athens, Greece, reported 90.7 per cent cases of chronic simple glaucoma and 9.3 per cent cases of congestive glaucoma, half of the latter cases being instances of acute glaucoma.

Rohner,[11] who studied 200 cases in Switzerland, found an almost equal distribution of cases of these two types—48 per cent cases of simple glaucoma and 52 per cent cases of congestive glaucoma. In the latter group, 54 per cent of the patients showed acute phenomena at one time or another. Rohner also gave a table of results of previous investigations (table 8).

TABLE 8.—*Number of Cases of Simple and of Congestive Glaucoma Found in Previous Investigations (Rohner)*

Investigator	Year	Simple Glaucoma	Inflammatory Glaucoma
Illig	1921	311	95
Kraus	1899	155	78
Schmidt-Rimpler	1908	195	157
Neuberger	1894	136	134
Schüssele	1899	253	553
Basallino	1906	81	266
Jacobsohn	1903	30	115

The considerable variance seen in these associated statistics from various countries is obvious and is undoubtedly due to the various criteria employed in classifying the congestive and noncongestive types. It would appear that with more intensive study of each case and with the employment of slit lamp examination more cases would fall into the group of cases of congestive glaucoma than have been reported. Moreover, such use of the corneal microscope in all cases would reveal changes, such as pigment dust on the iris or the capsule of the lens, cells in the anterior chamber, corneal deposits and other evidence of low grade uveitis, that would mark the case as one of the secondary type of glaucoma. Until such data are available, our cases must be classified, as they have been, merely on the basis of the history and the gross clinical findings.

9. Pillat, H.: Arch. f. Ophth. **139**:299, 1933.
10. Trantas, R.: Bull. et mém. Soc. franç. d'opht. **47**:277, 1934.
11. Rohner, M.: Schweiz. med. Wchnschr. **57**:780, 1927.

Age.—The factor of age is undoubtedly of the utmost importance in any statistical consideration of glaucoma; according to Elliot,[12] "Age takes precedence over all known causes of this disease." The distribution of cases during five decades of life for each diagnostic group and for the total group is shown in the tables and charts. The average age of the patients is 59.7 years for the females and 59.9 years for the males. According to census figures, the expectation of life for these persons is 14.1 years. This would indicate that one is dealing with a group of persons who will constitute a considerable proportion of the population for a substantial time and tends to emphasize the gravity of the situation in regard to glaucoma. Again, with the constantly increasing expectation of life and the survival of an older group of persons in the general population, the incidence of glaucoma will undoubtedly show an increase in the future.

In the series of 392 cases of congestive glaucoma, the greatest incidence, 36.2 per cent, was found for persons in the seventh decade of life. Similarly, in the group of cases of noncongestive glaucoma, in 37.4 per cent of the cases the condition occurred in the same period of life. Acute congestive glaucoma tends to occur somewhat earlier, the largest incidence, 35.4 per cent, being seen in the sixth decade of life, although chronic congestive glaucoma, like chronic simple glaucoma, appears most frequently in the seventh decade.

These figures agree substantially with those for Carvill's [2] series of patients, in 39.6 per cent of whom the condition occurred in the seventh decade, and with those for Haag's [7] series, in 31.8 per cent of whom it occurred at this time, although Priestley Smith's [13] series showed the greatest incidence by a small margin in the sixth decade, his figures being 29.3 per cent for the sixth decade and 29 per cent for the seventh decade. Weinstein's [8] series showed an incidence of 34.2 per cent in the age group of 55 to 64 in the cases of congestive glaucoma, although in his cases of noncongestive glaucoma the incidence for this age group tended to be somewhat lower, the greatest incidence being in the age group of 65 to 74. Pillat's [9] series in China showed an incidence of 34 per cent in the sixth decade. Nelander reported the greatest morbidity from glaucoma for persons in the age group of 70 to 79, Trantas for persons in the seventh decade of life and Rohner [11] for those in the second half of the sixth decade in all cases. In Rohner's cases inflammatory glaucoma tended to occur somewhat earlier than simple glaucoma.

Sex.—The influence of sex on the incidence of glaucoma has been accepted by practically all authors, the general consensus being that

12. Elliot, R. H.: Glaucoma, New York, Paul B. Hoeber, 1918.

13. Smith, Priestley: On the Pathology and Treatment of Glaucoma, London, J. & A. Churchill, 1891.

females suffer from primary glaucoma somewhat more frequently than males. Priestley Smith's [13] series included 56.9 per cent females, Haag's [7] 61 per cent and Carvill's [2] 48.8 per cent, including patients with the congestive and those with the noncongestive type. However, if all the groups in Carvill's series are considered, women outnumbered men in the group with congestive glaucoma, comprising 57 per cent of the patients, whereas 54 per cent of the patients with noncongestive glaucoma were men. Females comprised 60.7 per cent of Weinstein's [8] series of patients, most of whom had the congestive type of glaucoma, although his smaller series of patients with simple glaucoma included only 43.8 per cent females. In Pillat's [9] Chinese series, the females outnumbered the males 3 to 1. Nelander [1] has reported twice as many females as males in both cases of the simple type and those of the congestive type. In Trantas's [10] series, males slightly outnumbered females. Rohner [11] stated a predominance of female over male patients of 3 to 2;

TABLE 9.—*Incidence of Glaucoma in Males and Females as Found by Various Investigators (Rohner)*

Investigator	Males	Females
Neuberger	127	155
Jacobsohn	65	80
Schmidt-Rimpler	1,240	1,545
Mikulinska	5.34	12.54
Kraus	126	119
Illig	218	188

this was especially marked in acute congestive glaucoma. She quoted the following figures, taken from reports of previous investigators.

In our cases, taken as a whole, there was a slight preponderance in favor of females, who comprised 51.5 per cent of all the patients. In the group of patients with acute congestive glaucoma, somewhat contrary to most reports, the sexes were evenly divided—80 and 81. The number of females was more marked in the group with chronic congestive glaucoma, the incidence being 133 females and 98 males. Patients with chronic simple glaucoma included 514 females and 509 males—an almost equal distribution. If the data are analyzed in terms of congestive and noncongestive glaucoma, it is found that 54.6 per cent of the patients with congestive glaucoma and 50.3 per cent of those with the noncongestive type were females. Generally speaking, these figures correspond with those found in Carvill's [2] study of a fairly equivalent group in the Massachusetts Eye and Ear Infirmary.

Heredity.—The hereditary tendency of glaucoma has been stated by various writers. Definite information on this was not obtainable in practically all our cases, and no conclusions could be drawn. One patient, who gave us her ancestral history, stated that she had 2 sisters affected with glaucoma.

Race.—No information as to the racial origin of the patients was obtained as a routine at the time of their admission to the clinic.

However, such information was available for all patients admitted for operation during the years 1931-1935, inclusive, and this series included 590 cases. It was thought that some light might be thrown on the question as to the prevalence of glaucoma in the Jewish race, and these patients were grouped as Jewish and non-Jewish; 13.2 per cent of the patients with glaucoma hospitalized during this time were of the Jewish race. This figure slightly exceeds the percentage of Jews in the general population of Philadelphia, which is 12.2 per cent. We do not feel that the opinion as to the greater prevalence of glaucoma in Jews is strengthened by this finding. Carvill [2] found that the Jewish race was somewhat less susceptible to glaucoma than any of the other races that were represented in her material.

Previous Treatment.—Table 10 shows the number of patients who had had therapeutic treatment before applying for consultation or treatment at the hospital.

TABLE 10.—*Number of Patients Previously Treated*

	Type of Treatment		
Type of Glaucoma	Operative	Medical	Nonmedical
Congestive	55	45	17
Simple	180	76	22

Apparently a considerable number of patients with noncongestive glaucoma had had previous operative treatment. This is not surprising, in view of the fact that the Wills Hospital is often made use of by patients as a consulting agency, and many of this group came to the hospital as a last resort. Forty-nine patients had nonmedical supervision; they had generally applied to an optometrist, and many of these patients gave a history of frequent purchase of glasses over a short period.

Duration of Symptoms.—Table 11 summarizes the varying periods during which the patient had noticed symptoms, in the two diagnostic groups. Logically enough, patients with congestive glaucoma are more apt to apply for treatment early and patients with simple glaucoma, whose only symptom is visual loss, are more apt to defer opthalmologic consultation.

Refraction.—From the literature, it would appear that most authors agree that the glaucomatous eye is hyperopic. Priestley Smith's series of patients [13] showed the following states of refraction: hyperopia, 45.1 per cent; emmetropia, 40.9 per cent, and myopia, 14 per cent. Carvill's [2]

series showed: hyperopia, 64 per cent, and myopia, 29.2 per cent. This ratio is apparently more marked in congestive glaucoma than in the noncongestive type. Arnold Knapp [14] reported 32 cases of myopes with glaucoma, in all of whom the condition was the noncongestive type. In Weinstein's [8] series, 62 per cent of the patients were hyperopic and 14.6 per cent myopic. Rohner reported 67 cases of hyperopia, 9 cases of myopia and 130 cases of emmetropia. Our own results are shown in table 12.

The refraction was determined in only a comparatively small number of cases. Those patients whose refractive error exceeded 1 D. of

TABLE 11.—*Duration of Symptoms*

	Type of Glaucoma			
	Congestive (337 Cases)		Simple (1,003 Cases)	
Duration	No.	Percentage	No.	Percentage
Less than 6 months	161	47.70	211	21.03
Less than 12 months	32	9.49	132	13.16
2 years	33	9.79	152	15.15
3 years	18	5.34	278	27.71
4 years	6	1.78	90	8.97
5 years	30	8.90	84	8.37
5 + years	57	16.90	56	5.58

TABLE 12.—*Refractive State*

Type of Glaucoma	Number of Cases		Percentage
Congestive (total cases known, 144)	Hyperopia (> + 1 D.)	35	24.3
	Myopia (> — 1 D.)	20	13.9
	Emmetropia (+ — 1 D.)	46	31.9
	Mixed	43	29.9
Simple (total cases known, 165)	Hyperopia	68	41.2
	Myopia	24	14.5
	Emmetropia	49	29.7
	Mixed	24	14.5

hyperopia or myopia were considered hyperopic or myopic, respectively. Patients with a refractive state falling within the range of + 1 to — 1 D. were considered emmetropic. These figures correspond in general with the consensus as to the comparative frequency of refractive errors in persons with glaucoma.

Systemic Conditions.—The general medical status of the patient is undoubtedly of the utmost importance in evaluating a case of glaucoma. Some authors maintain that glaucoma is but a symptom of a systemic disease, which may be vascular, neurovegetative or metabolic. Unfortunately, data as to the general medical condition of the patients are

14. Knapp, Arnold: Tr. Am. Ophth. Soc. **23**:64, 1925.

lacking in most of our cases. The systemic conditions for those patients for whom this information could be secured and the number of patients are listed as follows:

Congestive Glaucoma

Syphilis	10
Arteriosclerosis	13
Hypertension	55
Diabetes	14
Nephritis	5
Uremia	1
Arthritis	4

Simple Glaucoma

Syphilis	9
Arteriosclerosis	8
Hypertension	45
Diabetes	23
Nephritis	8
Arthritis	5
Focal infection	26
Eczema	1
Myxedema	1
Pernicious anemia	1

The general impression from this tabulation would be that the incidence of syphilis is not more than one would expect in a sample of population such as this. Arteriosclerosis has been diagnosed in those cases in which the patients were referred to the medical clinic, as was hypertension. The reports furnished were practically the same as would be expected for any group of patients of corresponding age; that is, there was a heavy incidence of hypertension, arteriosclerosis, cardiovascular disease and cardiorenal disease.

Presence of Cataract.—Considering the interference with nutrition of the lens that occurs in glaucomatous eyes secondary to pathologic changes in the ciliary body and the angle of the anterior chamber, one would expect a greater prevalence of opacities of the lens in glaucomatous patients. Carvill [2] reported that 18 of her 49 patients between 60 and 70 years of age with congestive glaucoma had cataract (37 per cent) and 20 of her 60 patients in this seventh decade of life with noncongestive glaucoma had cataract (33 per cent). In her total series of 262 cases, 146 eyes had senile cataract. The figures for the incidence of changes in the lens in the various age groups are listed in table 13, and for comparison a table is given showing the incidence of the opacities of the lens in normal eyes as found by two authors (taken from Berens [15]).

15. Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, p. 714.

The figures for the development of cataract following operative procedures for the relief of glaucoma will be given in a later section.

Incidence of Seasonal Attacks of Acute Glaucoma.—It is a matter of interest, and perhaps of etiologic importance, to consider the season of the year during which attacks of acute congestive glaucoma developed in our patients. Weinstein [8] stated the belief that seasonal and climatic conditions are a factor in producing attacks of acute glaucoma, and in a series of 573 cases of this condition found the highest incidence to be during the winter months. Rohner [11] likewise found the

TABLE 13.—*Presence of Cataract*

Age Group	Congestive Glaucoma			Noncongestive Glaucoma		
	No. of Patients	No. with Opacities of the Lens	Percentage	No. of Patients	No. with Opacities of the Lens	Percentage
40-49	45	16	35.5	125	14	11.2
50-59	122	94	77.0	242	72	16.9
60-69	142	108	76.1	383	124	32.4
70-79	73	64	87.7	242	117	48.3
80-89	10	5	50.0	31	20	64.5
Total	392	287	73.2	1,023	347	33.9

TABLE 14.—*Percentage of Opacities of the Lens in Normal Eyes*

Age Group	Author	
	Anderson	Gradle
41-50	38.2	34.1
51-60	65.1	66.2
61-70	85.0	68.4
71 +	92.9	90.9

TABLE 15.—*Time of Year for Attacks of Acute Glaucoma*

Jan.	Feb.	March	April	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
17	13	15	10	12	9	9	8	8	10	13	17

greatest incidence during the months from November to February, the maximum being in November. She expressed the opinion that pupillary enlargement associated with the reduced amount of daylight, changes in the barometric pressure and frequent infections of the upper respiratory tract are explanatory causes. Pillat [9] regarded meteorologic conditions as having a good deal to do with fluctuations of the intra-ocular tension. German investigators reported the highest incidence of attacks of acute glaucoma during the winter. Moynant, in India, found them most frequent in June, when the solar radiation is at a minimum in that zone.

Table 15 shows the comparative frequency of attacks of acute glaucoma in 141 cases in our series. The tendency for attacks of acute

glaucoma to occur during the cooler months of the year is somewhat borne out by these findings.

Stage When Patient Was Seen.—In our series of patients with congestive glaucoma, only 9 were seen early, with one eye apparently normal and beginning glaucoma in the other eye. Twenty-eight patients first appeared at the clinic with an early stage of glaucoma in both eyes. In 48 cases the condition was well advanced in one eye without involvement of the other, and in 51 patients the condition was in the late stage in one eye and still unilateral. Sixty patients were first seen with

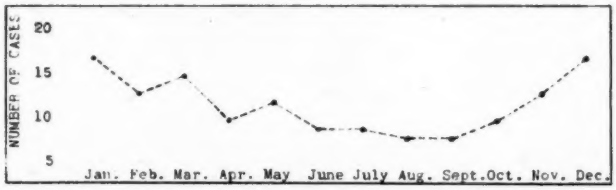

Chart 3.—Frequency of attacks of acute glaucoma in 141 cases in the months of the year. The average figures for the ten year period 1926-1935 are used.

TABLE 16.—*Stage at Which Patients with Unilateral, and Those with Bilateral, Congestive Glaucoma Were Seen*

Age Group	Unilateral (133 Cases, 41.9%)			Bilateral (196 Cases, 58.1%)			
	Early	Moderately Advanced	Late	Early	Moderately Advanced	Late	Absolute
40-49	2	10	2	8	10	6	6
50-59	3	12	13	6	22	34	8
60-69	4	18	24	8	24	38	12
70-79	..	8	11	6	3	24	18
80-89	..	..	1	..	1	6	4
Total	9	48	51	28	60	108	48
Percentage	2.6	14.5	22.4	8.3	17.8	32	14.5

both eyes involved to a well advanced degree. The largest number of patients, however, came to the clinic with late involvement of both eyes —108 persons; 48 patients had bilateral absolute glaucoma at the time of their first admission; as a group, 41.9 per cent of the patients with congestive glaucoma were first seen when the condition was unilateral, and 58.1 per cent were seen when the condition was bilateral, as shown in table 16.

Analyzing the cases of simple glaucoma in a similar way, we found that 78 patients had early unilateral simple glaucoma, 199 moderately advanced simple glaucoma and 219 late unilateral simple glaucoma. Early bilateral simple glaucoma was present in 88, moderately advanced simple glaucoma in 167 and late simple glaucoma in 227; 70 patients

did not come to the clinic until the condition had reached an absolute stage in each eye; 784 eyes had vision reduced to 6/60 when the patients first presented themselves. This comparatively large figure is a commentary on the state of knowledge of glaucoma among the general population. It is true that many of these patients came from rural districts where no facilities for diagnosis and treatment were available. As a group, 50.7 per cent of the patients with chronic simple glaucoma showed unilateral involvement, and 49.3 per cent bilateral involvement at the time of their first admission.

TABLE 17.—*Stage at Which Patients with Unilateral, and Those with Bilateral, Simple Glaucoma Were Seen*

	Unilateral (496 Cases, 50.7%)			Bilateral (482 Cases, 49.3%)			
Age Group	Early	Moderately Advanced	Late	Early	Moderately Advanced	Late	Absolute
20-29	..	..	..	..	..	..	..
30-39	..	..	..	..	..	..	..
40-49	23	30	35	27	44	39	6
50-59	20	58	56	25	38	59	20
60-69	22	72	59	23	56	79	21
70-79	13	36	58	12	29	45	14
80-89	..	3	11	1	..	5	9
Total.....	78	199	219	88	167	227	70
Percentage of 978 cases....	7.9	20.3	21.8	8.9	17.0	23.2	

FOLLOW-UP STUDY

With the exclusion of 768 patients who resided outside of Philadelphia and with whom no attempt at contact was made, the follow-up and field aspect of the study concerns itself with 1,108 patients, residents of Philadelphia, who at various times during the period from 1926 to 1935 had appeared at the clinics of the Wills Hospital and whose condition was diagnosed as glaucomatous (including patients with secondary glaucoma).

Letters were addressed to the homes of these 1,108 patients, requesting them to appear at a stated hour at the hospital for further examination of their ocular condition; 573, or 56.8 per cent, of these letters were returned by the post-office as undeliverable. No further attempt was made to contact these persons.

It was assumed that the remaining 535 letters had been delivered and that the patients had been notified of the hospital's request. A certain number of persons began to appear for examination. After waiting a reasonable period, field workers (registered nurses) were sent to interview those patients who had not appeared and to assist them in reaching the hospital. Of this group, 290 patients were contacted in their homes by nurses; 245 persons could not be located, although in certain instances information concerning the patient was obtained from the

neighbors, i. e., data as to death, the condition of eyes and other information. Two hundred and sixty-four patients in all actually reported to the hospital and were seen.

On the basis of 535 possible contacts, this is a field contact efficiency of 49.3 per cent; on the basis of 1,108 patients to be investigated, the figure for contact efficiency is 26.2 per cent. Considering the age range of the persons concerned and the heterogeneity of the group, the number of contacts made may be considered as not unsatisfactory. Table 18 outlines these data.

It was definitely determined by field workers that 61 patients had died, 14 were being treated at other hospitals, 6 were confined to insti-

TABLE 18.—*Data for Patients Considered in This Study*

	Patients Residing in Philadelphia			
		Letters Apparently Delivered; Field Workers Sent Out (535 Cases)		
Year	Letters Returned by Post Office	Patients Contacted	Patients Not Contacted	Patients Actually Reporting
1926	40	40	11	5
1927	101	36	13	9
1928	79	25	25	9
1929	98	26	51	28
1930	41	48	25	47
1931	45	23	25	42
1932	39	21	23	24
1933	78	23	28	25
1934	42	26	29	43
1935	30	22	15	32
	573	290	245	264

Total number of patients	
Residing in Philadelphia	1,108
Residing outside of Philadelphia	768
Total number of patients reporting	264
49.3% of 535 patients apparently still in Philadelphia	
26.2% or 1,108 original patients	

tutions for the aged or blind, 20 were under the private care of ophthalmologists and 14 were recipients of pensions for the blind. Accordingly, the whereabouts of 115 patients was determined in addition to that of the group who reported for follow-up study.

TREATMENT

Consideration of the treatment employed in these cases is complicated by the fact that patients were under the management of nine different surgeons incumbent in the hospital at various times over the period of ten years; varying procedures were thus employed, and varying interpretations may be placed on the results. However, in general, it may be stated that the procedure for each patient was about as follows: On the first admission to the clinic the vision was recorded with and without glasses, an external examination of the eyes and an examination of the fundi were made, and the intra-ocular tension was

measured by means of a tonometer (the Schiötz instrument was used as a routine, but there was limited use of the McLean instrument during 1926 to 1929); whenever possible, the visual fields were obtained, and medical examination was done when indicated, as were laboratory studies. The subsequent treatment was determined by the nature of the case, and no uniform statements can be made as to the routine management of the various types of glaucoma.

The treatment falls into two groups: (1) operative and (2) non-operative, or treatment with miotics. Operative procedures were limited to the following: (1) cyclodialysis; (2) paracentesis and posterior sclerotomy, considered as temporary alleviative measures; (3) basal iridectomy; (4) the Elliot trephine operation, and (5) the modified

TABLE 19.—*Operations Performed in the Three Diagnostic Groups*

Operation	Acute Congestive	Chronic Congestive	Simple	Total
Cyclodialysis	9	10	36	55
Paracentesis	10	6	2	18
Sclerotomy	12	25	8	45
Iridectomy	95	109	148	352
Elliot trephine operation	46	64	268	378
Lagrange operation	5	20	27	52
Operations				900
Enucleation				
Acute congestive glaucoma				12
Chronic congestive glaucoma				28
Simple glaucoma				32
Total				72
Extraction of cataract				
Congestive glaucoma				26
Noncongestive glaucoma				42
Total				68

Lagrange operation. A small group of patients were also seen who received no treatment over a period of years and can thus serve, in a sense, as a control series.

Table 19 enumerates the various operations performed in the cases of the three diagnostic groups of glaucoma. In all, 900 operative procedures were performed to reduce the intra-ocular tension. In the entire series, 72 enucleations were done and 68 operations for extraction of cataract performed on glaucomatous patients.

Table 20 indicates in more detailed fashion the operative procedures and the results evaluated in terms of reduction of the intra-ocular pressure. Those cases in which an intra-ocular tension below 25 mm. of mercury was maintained during the postoperative period of observation, which ranged from two to nine years, are noted under the column "Tension Reduced." It is true that a considerable number of these patients continued to use miotics during this period, but in cases

in which no secondary operation was considered necessary the primary operative result for the purposes of this survey is evaluated as satisfactory. All the figures are given in terms of individual eyes, except in the instances in which only miotics were employed, which are considered as cases. Postoperative complications, including hemorrhage of any significant amount, infection, the development of cataract and choroidal or retinal detachment, are noted for each type of operation under each variety of glaucoma.

TABLE 20.—*Results of Operations on Eyes in the Three Diagnostic Groups Evaluated in Terms of Reduction of the Intra-Ocular Tension*

	Acute Congestive			Chronic Congestive			Simple		
Treatment	Number	Tension Reduced	Tension Not Reduced	Number	Tension Reduced	Tension Not Reduced	Number	Tension Reduced	Tension Not Reduced
Cyclodialysis	9	3	4	10	..	5	36	14	18
Complications (postop.)	Hemorrhage, 2			Hemorrhage, 1			Cataract, 1; hemorrhage, 2		
Paracentesis	10	6	2	6	4	2	2	..	..
Sclerotomy	12	8	1	25	14	4	8	6	..
Complications (postop.)	.....			Hemorrhage, 1					
Iridectomy	95	40	15	109	52	11	148	60	39
Complications (postop.)	Cataract, 2; choroidal detachment, 1; infection, 1; loss of vitreous, 1			Hemorrhage, 2; cataract, 2; retinal detachment, 1			Hemorrhage, 3		
Elliot trephine operation	46	24	6	64	29	12	278	165	59
Complications (postop.)	Cataract, 1; choroidal detachment, 1			Cataract, 2; infection, 1			Cataract, 9; hemorrhage, 8		
Modified Lagrange operation	5	2	3	20	6	9	27	17	7
Complications (postop.)	.....			Infection, 1					
Miotics (cases)	23	4	1	69	6	10	257	74	78

Table 21 expresses in percentage form the tension-reducing efficiency of each operation for each type of glaucoma. In this table the operations paracentesis and sclerotomy are not considered, since it is felt that they are but temporary expedients and should not be included in the final disposition of the case.

It would seem hazardous to evaluate the worth of an operative procedure in these terms, but this has been done by various authors, a few of whom can be cited for comparison. Arnold Knapp,[16] in treating 200 patients with chronic glaucoma, obtained successful reduction of

16. Knapp, Arnold: Operative Treatment of Chronic Glaucoma: Report of Two Hundred Successful Operations, Arch. Ophth. **10**:298 (Sept.) 1933.

tension in 5 of 12 by iridectomy, in 60 of 80 by the Elliot trephine operation, in 85 of 95 by the Lagrange operation and in 5 of 8 by cyclodialysis. Etienne [17] obtained reduced tension in 114 of 187 cases; he employed the trephining operation in 89 and the Lagrange operation in 29. Kotljarevskaja obtained reduced tension in 97 per cent of cases in which the Elliot trephining operation was done. Philippow, in a series of 54 cases, after cyclodialysis obtained reduced tension in 98.1 per cent. Bothman and Blaess [18] used the Elliot trephine operation in 70 cases and obtained a reduction of tension in 98.2 per cent.

Our own series, which includes instances of all stages, would seem to indicate that the Elliot operation has comparatively the highest efficiency in simple glaucoma. As a whole, cyclodialysis does not appear to be of great value, although it must be remembered that in many of the cases in which this operation was utilized the condition was

TABLE 21.—*Tension-Reducing Efficiency of Operations for Each Type of Glaucoma*

	Acute Congestive			Chronic Congestive			Simple		
Treatment	No. of Eyes Followed	Tension Reduced	Percentage	No. of Eyes Followed	Tension Reduced	Percentage	No. of Eyes Followed	Tension Reduced	Percentage
Cyclodialysis	7	3	43	5	0	0	32	14	44
Iridectomy	55	40	73	63	52	83	99	60	61
Elliot trephine operation	30	24	80	41	29	71	224	165	74
Modified Lagrange operation	5	2	40	15	6	40	24	17	71
Miotics (cases)	5	4	80	16	6	38	152	74	49

advanced and complicated. Basal iridectomy, as generally accepted, is more efficient in the congestive types of glaucoma, particularly when employed early, before adhesion of the root of the iris. The series in which the Lagrange operation was performed is too small to permit of any conclusions. Continued treatment with miotics in chronic simple glaucoma was efficient in only 49 per cent of the cases. This is in agreement with the thought that chronic simple glaucoma is essentially a surgical disease.

ANALYSIS OF SMALLER FOLLOW-UP SERIES

In a smaller group of cases, in which the patients appeared in person for follow-up study and could be examined somewhat more carefully, it was possible to draw conclusions that are probably more authentic.

Congestive Glaucoma.—The cases of fifty eyes that were affected with congestive glaucoma in which the patients had been followed from four to nine years are analyzed as follows with respect to the operative procedures and the visual results (table 22).

17. Etienne, quoted by Bothman and Blaess.[18]

18. Bothman, L., and Blaess, N. J.: Am. J. Ophth. **19**:1072, 1936.

Cyclodialysis was performed in 3 cases as a primary operation and was effective in reducing tension in 1 case. However, the vision deteriorated, and in all the patients absolute glaucoma was the outcome. Cyclodialysis was performed in 2 cases as a secondary operation following unsuccessful iridectomy and the Lagrange operation, respectively; the tension was not reduced in either case.

Iridectomy: There were 15 cases in which iridectomy was performed as a primary operation; the tension was reduced in 10 eyes and not reduced in 5; the eventual visual result, as judged by visual acuity and the fields, was good in only 5 eyes. In 2 cases the condition ended in the absolute stage.

Elliot Trephine Operation: This procedure was performed as a primary operation on 11 eyes and was successful in reducing the tension in 8. In 6 cases the visual efficiency was maintained as a result

TABLE 22.—*Outcome of Operations in Smaller Series of One Hundred and Sixty-Five Eyes with Congestive or Noncongestive Glaucoma*

Treatment	Number	Tension Reduced	Tension Not Reduced	Operation Successful, Percentage of Eyes	Complications
Cyclodialysis	6	1	5	16	
Iridectomy	30	19	11	63	Cataract, 1
Elliot trephine operation	59	48	11	81	Cataract, 4
Lagrange operation	10	7	3	70	
Miotics	60	42	18	70	

of the operation. The Elliot trephine operation was performed on 1 eye as a secondary operation following a previous iridectomy and was not successful. Three patients showed opacities of the lens subsequent to the Elliot trephine operation.

Lagrange Operation: This procedure was performed on 5 eyes; the tension was reduced in 4, and vision was maintained in 4; the fifth eye showed low grade postoperative iridocyclitis.

Treatment with Miotics: This treatment was continued for 13 eyes (usually the mates of eyes that had been operated on). The reduced tension was maintained in 9 cases; vision was maintained in only 7 eyes.

Comment.—Two instances may be cited for the purpose of comparing operative and nonoperative treatment. In one patient an Elliot trephine operation on the right eye was successful, whereas treatment with miotics of the left eye gave a poor result. On the other hand, in another patient a Lagrange operation on the right eye and therapy with miotics for the left eye were equally effective.

As a control instance there may be cited that of a patient seen three years previously with a condition diagnosed as glaucomatous who was frightened away and neglected to obtain any treatment.

When he was called back to the hospital for the follow-up study it was found that vision had deteriorated in the affected eye from 6/21 to perception of light.

Noncongestive Glaucoma.—A second smaller group of eyes affected with chronic simple glaucoma was also available for better analysis. This group consisted of 115 eyes. When first seen, 44 of these eyes had vision reduced below 3/60. The detailed analysis with respect to the operative procedures is as follows:

Cyclodialysis (1 case): The tension was not reduced; cataract developed, which was extracted, with no improvement in vision.

Iridectomy (15 operations): The tension was reduced in 9 eyes, in which vision was well maintained. One cataract developed in this series, which was successfully removed.

Elliot Trephine Operation (47 eyes): The tension was reduced in 43 eyes and vision was maintained in 32; operation for cataract was performed on 3 eyes in this series.

Lagrange Operation (5 cases): The tension was reduced in 3 eyes; however, in only 1 of the cases was there a good visual result.

Treatment with Miotics: Forty-seven eyes were treated with miotics; the tension was reduced in 33, in 30 of which vision was maintained.

Control Group.—Ten patients reported who had had no treatment for from two to nine years. These persons had been seen only once or twice during their original period of observation at the clinic and for varying reasons had neglected to obtain any further medical supervision. Several had been frightened away when operation was originally advised; others had expressed dissatisfaction with their original treatment and had discontinued their visits to the hospital. In these 10 patients, the condition in 11 eyes deteriorated to the point of absolute glaucoma. Thus, 1 patient, who was first seen in 1927 and who received no treatment, with an initial vision of 6/21 in the right eye and perception of light in the left, when seen in 1936 had a small temporal field in the right eye and no perception of light in the left. In another patient vision had deteriorated during the period of 1930 to 1936 from 6/60 to 6/30 to no perception of light and perception of light, respectively. On the other hand, 1 person who had an original tension of 28 mm. four years previously was found to have the same tension, the same vision and the same fields on follow-up examination.

It is apparent, however, from the findings for these control patients, that untreated glaucoma usually progresses to inevitable blindness and that ophthalmologic treatment, comparatively inefficient as it may be, is of definite value.

SUMMARY AND CONCLUSIONS

The records for 1,876 patients with glaucoma seen at the Wills Hospital during the ten years from 1926 to 1935 were examined and analyzed. This represents an incidence of glaucoma of 0.78 per cent in the patients with ocular conditions.

In the group, 413 cases of secondary glaucoma were found—22 per cent of the total series of cases.

Trauma and syphilis were the two most prominent factors in the causation of secondary glaucoma.

The development of a secondary rise in tension in any given ocular lesion is of grave significance; in 34 per cent of the cases of secondary glaucoma the condition ended in serious visual impairment.

Twenty-eight cases of congenital, and 20 cases of juvenile, glaucoma are reported.

One thousand, four hundred and fifteen cases of primary glaucoma are reported, 27.7 per cent cases of the congestive type and 72.3 per cent cases of the noncongestive type. These are analyzed on the basis of age, sex, refractive error, race, previous treatment, duration of symptoms and other factors.

The average age of the males in the series was 59.9 years, and that of the females, 59.7 years.

There appears to be no evidence for the theory that the Jewish race is more susceptible to glaucoma than other races.

There is evidence for the belief that acute attacks of glaucoma tend to occur during periods of colder, more unstable weather.

The results of the follow-up study and field work carried out among the patients living in Philadelphia are reported.

The results of treatment for the various types of glaucoma are presented from the standpoint of reduction of the intra-ocular tension during the period of observation, ranging from two to ten years.

It would appear that the Elliot trephine operation is the most efficient form of operative treatment.

A small control group of patients receiving no treatment is presented to show the ultimate ocular damage in untreated patients.

From the standpoint of public health, the most significant finding is the fact that 784 eyes had vision already reduced below 6/60 when the patients first presented themselves at the hospital. This is a commentary on the state of ignorance of the early signs and symptoms of glaucoma existent among patients, to some extent among family physicians and certainly among nonmedical ocular practitioners.

ACCOMMODATION AND THE AUTONOMIC NERVOUS SYSTEM

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The rôle of the sympathetic nervous system in accommodation has been variously assumed and denied, but its active participation seems necessary to explain certain clinical and experimental phenomena to be presented in this paper. It would appear that the sympathetic system tends to adapt the eye for relatively distant objects and as such opposes the parasympathetic system, which tends to adapt the eye for relatively near objects.[1]

The mechanism whereby the sympathetic system effects this distance adjustment is not obvious. It is suggested that the radial fibers of the ciliary muscle are innervated by the sympathetic system and on contraction exert a pull on the zonule which flattens the lens. They would be opposed, of course, by the circular fibers which on contraction decrease the circumference of the ciliary ring and allow greater curvature of the lens as postulated in Fincham's [2] modification of the Helmholtz [3] theory.

Although this concept occurred to me independently it has been suggested in part by previous authors. Helmholtz [3] in 1855 considered the possibility of a dual nature of the ciliary muscle, but he discredited the idea for two reasons. In the first place, he thought the muscle fibers were too intertwined to allow of discrete actions. Second, he noted that atropine paralyzed the ciliary muscle and could do so without changing the refractive condition of the eye. As for the first objection, it is known that in other organs, such as the gastro-intestinal tract and the uterus, one finds intimate anatomic connections between antago-

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1. I am aware that either a flattening or an increased curvature of the lens may be induced by factors other than distance, e. g., lenses. However, for convenience I shall use the terms "distance accommodation" and "near accommodation," as relative distance is customarily associated with the stimulus of accommodation, whatever the nature of that stimulus may be.

2. Fincham, E. F.: The Mechanism of Accommodation, Brit. J. Ophth., supp. 8, 1937, p. 5.

3. Helmholtz, H.: Ueber die Accommodation des Auges, Arch. f. Ophth. (pt. 2) **1**:1, 1855.

nistically acting groups of muscles, and apparently this does not interfere with their separate functions. As for the second objection, there is no reason to believe that atropine does paralyze the entire ciliary muscle. It is known that atropine paralyzes only such smooth muscle as is innervated by the parasympathetic system. One would not expect, therefore, the refractive condition of the eye to be changed under the influence of atropine except so far as the parasympathetic system affects it, that is, in accommodating for near and in releasing what antagonism the parasympathetic system tonically offers to the sympathetic system. The latter effect, of course, is maximal in hyperopia, in which atropinization does alter the manifest refractive condition, and minimal in myopia, in which atropinization alters the manifest condition little, if at all.

Henke [4] in 1860 postulated the theory that a ciliary muscle has two components, the circular fibers functioning for near accommodation and the longitudinal fibers for distance. Warlomont [5] (1875) made an anatomic study of the ciliary muscle and reiterated Henke's hypothesis, pointing out the fallacy of the terms "passive" and "negative" accommodation for the distance adjustment. Morat and Doyon [6] in 1891 were apparently the first to suggest that the sympathetic system effected distance adaptation and the parasympathetic system near adaptation. Henderson [7] in 1926, apparently unaware of Morat and Doyon's thesis, proposed innervation of the ciliary muscle by both the sympathetic and the parasympathetic system, stating, "Hitherto the ciliary muscle has been regarded as being actuated by one nerve only, the third nerve, in spite of the fact already noted that smooth muscle is always provided with two sets of nerves." He, like myself, was of the erroneous opinion that he was the first to postulate the existence of the dual nerve supply. He assumed that the sympathetically controlled radial fibers were "postural" and served to "fix" accommodation induced by the third nerve. This was obviously an effort to reconcile the action of the ciliary muscle with Sherrington's and Hunter's theories of the control of posture by sympathetic nerves.

Poos [8] in 1928 made the highly interesting observation that after lesions of the cervical portion of the sympathetic system epinephrine hydrochloride instilled into the eye decreased accommodation, whereas it affected normal eyes practically not at all. This is significant in

4. Henke, W.: Der Mechanismus der Accommodation für Nähe und Ferne, Arch. f. Ophth. (pt. 2) **6**:53, 1860.

5. Warlomont, E.: Le muscle ciliare, Ann. d'ocul. **73**:195, 1875.

6. Morat, J. P., and Doyon, M.: Le grand sympathique, nerf de l'accommodation pour la vision des objets éloignés, Ann. d'ocul. **106**:28, 1891.

7. Henderson, T.: The Anatomy and Physiology of Accommodation in Mammalia, Tr. Ophth. Soc. U. Kingdom **46**:300, 1926.

8. Poos, F.: Zur Frage der sympathischen Innervation des Ziliarmuskels und Ihrer Bedeutung für die Akkommodation, Klin. Monatsbl. f. Augenh. **80**:749, 1928.

view of the known sensitization of structures innervated by the sympathetic system to epinephrine after lesions of the pathways of the sympathetic system. Hudelo [9] in 1930 stated that the assumption of the existence of a ciliary muscle with innervation by antagonistic systems is necessary for an understanding of the rapidly occurring refractive changes seen in diabetes and other diseases. Luedde [10] in 1932, on the contrary, stated: "According to all available facts, it seems unlikely that the mechanical readjustments of the tissues incident to accommodation are modified by sympathetic enervation."

Byrne [11] in 1933, using an instrument which he called an oculomanometer, showed by rather elaborate experiments that stimulation of the sympathetic system induced a flattening of the lens and stimulation of the parasympathetic system induced an increased curvature of the lens. An oculomanometer as devised by him consists of "a balance with a long and a short arm, the former carrying the writing point, whilst the latter carries a rigid, adjustable, vertical rod which descends from, and at right angles to, the end of the short arm . . . until its free end rests upon . . . the center of the lens," a small trephine hole having been made in the cornea. He also showed that both the increase and the decrease in convexity, respectively, could be sensitized to the action of pilocarpine and epinephrine by prior section of the respective autonomic nerves. Byrne further assumed that the radial portions of the ciliary muscle are innervated by the sympathetic system and the circular portions are innervated by the parasympathetic system. On studying Byrne's report I was impressed by the identity of my conclusions with his. However, there are several potential objections to Byrne's experiments. They were performed on animals and under obviously unnatural conditions—a hole in the cornea, an evacuated anterior chamber, and a recorder on the anterior surface of the lens, which might indicate nothing more than a gross displacement of the lens. My approach, on the contrary, has been through clinical observations on the intact eye, and I have come to the same conclusions. I believe that I have substantiated by studies on man Byrne's conclusions based on experiments on animals.

Nicolai [12] in 1935 assumed a dual action of the ciliary muscle, with an antagonism between the radial and circular portions, and suggested

9. Hudelo, A.: Mécanisme de l'accommodation et myopie diabétique, Arch. d'opht. **47**:70, 1930.

10. Luedde, W. H.: The Mechanism of Accommodation, Facts and Fancies, Arch. Ophth. **7**:40 (Jan.) 1932.

11. Byrne, J. G.: Studies on the Physiology of the Eye, London, H. K. Lewis & Co., Ltd., 1933.

12. Nicolai, C.: Der Mechanismus der Akkommodation, Klin. Monatsbl. f. Augenh. **94**:617, 1935.

on a priori grounds that the two components might be innervated by the sympathetic and the parasympathetic system. He presented only hypothetic evidence to corroborate his assumption. Strebel [13] in 1936 considered a dually acting ciliary muscle, but rejected this concept, as did Helmholtz, largely on the basis of the results of atropinization (and also because of his inability to show a biphasic action current of the ciliary muscle). He stated that were there a muscular apparatus the contraction of which accommodates the eye for distance, one would have to make the unlikely assumption that it is not paralyzed by atropine but rather that atropine brings about a cramplike contraction. This is exactly what I propose. Why he believed that this is unlikely he did not state, but certainly this contractive effect of atropine has its counterpart in every other structure innervated by a dual system, e. g., the iris, in which atropinization allows a cramplike contraction of the muscle innervated by the sympathetic system.

THEORETICAL CONSIDERATIONS

a. Evidence for Dual Innervation of the Eye by Analogy.—Those internal organs the functions of which are regulated by involuntary muscle are innervated by a dual system, the two components of which are mutually antagonistic. These two components are, of course, the sympathetic and parasympathetic nervous systems. In the case of the heart, for example, impulses from the sympathetic division are cardiac accelerators, while those emanating from the parasympathetic division are cardiac depressors. The same antagonism holds for other organs. In some cases the sympathetic system is excitatory, while in other cases the parasympathetic system is excitatory. In the gastro-intestinal tract, unlike the heart, it is the parasympathetic system which excites motility, while the sympathetic system has the reverse effect. Thanks to the work of Cannon [14] and others, the dominance of the one or the other system has been shown to have biologic utility. The sympathetic syndrome is invoked in response to environmental emergencies and manifests itself in the protective reactions of fear, anger and rage. When the environment is controlled as regarding temperature and other factors, a completely sympathectomized animal may live a fairly normal life but would be unable to adjust itself to the exigencies of a variable environment. The parasympathetic system, on the other hand, functions chiefly when no emergency is imminent.

13. Strebel, J.: Ueber die Mechanik des Akkommodationsvorganges und die Wirkungsweise der drei Fasersystem des Ciliarmuskels zur Widerlegung der Diskontinuitäts-theorien, Klin. Monatsbl. f. Augenh. **95**:235, 1935.

14. Cannon, W. B.: Bodily Changes in Pain, Hunger, Fear and Rage, New York, D. Appleton & Company, 1920.

So far, therefore, as the eye shows this antagonistic control of its autonomic functions, it is no exception, for analogous structures elsewhere in the body show a dual nerve supply.

As for the more immediate analogy between the ciliary body and the iris, it is known that accommodation is accompanied by miosis. Contraction of that portion of the ciliary muscle causing accommodation for near is accompanied by contraction of the sphincter of the iris. If the ciliary muscle is thought of as a single unit and under the influence of only one branch of the autonomic system, a similar mechanism would be expected in the iris. Such was conceived to be the case prior to the recognition of the dilator muscle by Langley and Anderson [15] in 1892.[16] Theretofore, the pupil was generally believed to be under the sole control of the sphincter, which produced miosis through contraction and mydriasis through inhibition. Whatever part inhibition of the sphincter plays, it is now known that the positive force of mydriasis is contraction of the sympathetic dilator muscle, and I believe that a similar positive force accommodating the eye for distance is present in the radial fibers. The radial fibers would seem to me to be synergistic with the dilator pupillae, just as the circular portion is synergistic with the sphincter pupillae. Mydriasis is associated with distance, just as miosis is with near accommodation.

Poos [8] emphasized the analogy between the innervation of the ciliary muscle by the sympathetic system and that of the iris on pharmacologic grounds. He noted that cocaine affects the normal iris and normal ciliary body but has practically no effect on either after section of the cervical portion of the sympathetic chain, whereas instillations of dilute epinephrine hydrochloride have practically no effect on the normal iris or ciliary body but have profound effects on both after lesions of the cervical portion of the sympathetic system.

By analogy, therefore, with other smooth muscle, and especially with that of the iris, the ciliary muscle may be expected to have a double innervation. Many of the discrepancies which formerly applied to the iris also apply to the ciliary muscle unless such a double function is assumed.

b. Anatomicophysiologic Factors.—The muscle fibers of the ciliary body are generally divided into three groups according to their direction: (1) circular fibers, which are concentric with the lens, forming a con-

15. Langley, J. H., and Anderson, H. K.: On the Mechanism of the Movements of the Iris, J. Physiol. **13**:554, 1892.

16. It is of interest to note that one of Langley and Anderson's objections to the alleged control of the pupil purely by the parasympathetic system was the partial miosis in Horner's syndrome. One of my major objections to considering the ciliary muscle as controlled purely by the parasympathetic system is, similarly, the otherwise inexplicable accommodative phenomena associated with Horner's syndrome.

tinuous and endless muscle belly, sphincter-fashion; (2) radial fibers, originating at the immobile scleral spur and radiating fanwise toward the surface of the ciliary body, and (3) meridional fibers, which also originate at the scleral spur and which extend toward the choroid. The groups, especially the circular and radial, are closely intertwined, and unless one has a meridional cross-section of the eye it may be impossible to differentiate the fibers.

So much for the ciliary muscle. The zonular fibers, through which the muscle acts, arise over the entire surface of the ciliary body, except for the apexes of the processes, and are attached chiefly to the para-equatorial portions of the lens. There is some dispute as to the origin of the individual fibers, but evidence to date would seem to be against the earlier impression that the fibers attach directly to the epithelium of the ciliary body. Wolfrum [17] has traced the fibers through the epithelial layer into the subepithelial tissue. As the fibers approach the lens they are arranged in a concentric fashion on the anterior and posterior capsules, each fiber having its insertion by innumerable filaments which are spread over a wide base. The anterior capsule receives about three fifths of all the zonular fibers.

On an anatomic basis, it is apparent that the sphincter-like circular fibers will tend, on contraction, to release the tension on the zonule and allow the lens to increase its curvature. The action of the radial fibers, however, is not so simple, as these fibers form an obtuse angle with the zonule. Their action would be twofold, a forward and an outward pull on the ciliary end of the zonule, the former effect being maximal on the posterior muscle fibers and the latter pull maximal on the anterior fibers. It will be remembered that the bulk of the radial fibers are placed anteriorly. The anterior displacement would in itself tend to release the tension on the zonule were it not compensated and, in my opinion overbalanced by the outward movement which increases the zonular tension and flattens the lens. This anterior displacement may have the effect of decreasing the angle between the radial fibers and the zonular fibers and thereby rendering the resultant outward force more nearly tangential to the anterior surface of the lens. The anterior surface is, of course, the place where the main accommodative changes take place. It appears to me, therefore, that the radial muscle fibers comprise that portion of the ciliary muscle which accommodates the lens for distance. At least positive distance accommodation must be accounted for in some fashion even if the specific mechanism here suggested does not meet with the reader's full agreement.

17. Wolfrum, M.: Ueber den Ursprung und Ansatz der Zonulafasern im menschlichen Auge, Arch. f. Ophth. **69**:145, 1908.

I am not yet in a position to account for the function of the meridional fibers. It may be that they take no part in the accommodative act but rather function in the control of the intra-ocular tension through their action on the choroidal and ciliary vessels, similar to the action of smooth muscle on erectile tissue elsewhere. Nor am I attempting any detailed explanation of the function of the most posterior zonular fibers, which, running from the posterior surface of the lens to the posterior portions of the ciliary body, are connected with the vitreous by many fine fibrillae. It is my opinion at present, however, that these are more concerned with delimiting the vitreous than with the function of accommodation.

Unfortunately, an unwarranted terminology has been applied to accommodation which confuses the issue. Thus one speaks of accommodation as a property of changing one's focus from a distant object to a nearer object and calls the reverse relaxation of accommodation. If, as I believe, the radial fibers adapt one's focus for relatively distant objects and the circular fibers adapt one's focus for near objects, the one is just as much an accommodative function as the other. They exist in a state of reciprocity similar to that of the dilator and sphincter muscles of the pupil, and it would be simpler if there were names for them similar to mydriasis and miosis, but since such names are lacking I am referring to the two as accommodation for distance and accommodation for near, respectively.

On physiologic grounds, the relatively greater speed with which one may accommodate for distance than for near would seem to indicate that accommodation is not purely a function of the parasympathetic system. It is generally believed that smooth muscle of the autonomic nervous system is activated by some intermediary chemical, which in the case of the sympathetic system is sympathin and in the case of the parasympathetic system is acetylcholine. Were the ciliary muscle under the sole influence of the parasympathetic system, one would expect a latent period in the relaxation of the muscle for focusing on distant objects while acetylcholine was being decomposed. Although acetylcholine is very unstable, its decomposition must be slower than its generation, else no free acetylcholine would ever be present. This would mean that near accommodation would always be more rapid than distance accommodation. Subjectively, some persons find the reverse to be the case, distance accommodation being more rapid than near accommodation. Robertson,[18] using the tachistoscope of Ferree and Rand, found that those having a more rapid distance accommodation were in the minority, but the fact that any reliable group of observers have a

18. Robertson, C. J.: Measurement of Speed of Adjustment of Eye to Near and Far Vision, Arch. Ophth. **14**:82 (July) 1935.

more rapid distance accommodation than others is evidence that the phenomenon is not explicable on the basis solely of the generation of the drug of the parasympathetic system, acetylcholine.

c. Evidence for Dual Innervation from the Point of View of Comparative Anatomy.—Herbivora have practically a negligible accommodation for near, carnivora have only a moderately well developed accommodative function, while it is not until one comes to the primates that accommodation exists as one knows it in man. Thus near accommodation in the horse, cow and rabbit is practically nil. In the dog and cat it is no more than from 2 to 4 D., while in apes it is from 10 to 15 D., and in man it may be considerably more.

The biologic reason for this difference in accommodative amplitude is most succinctly expressed in the following quotation from Treacher Collins:[19]

"The herbivorous terrestrial mammals, whose safety and survival depend on their swiftness of flight from danger . . . grasp their food directly with their mouths, finding that which is suitable more by smell and by the touch of their acutely sensitive snouts than by their sight. Their visual organs must be adapted for . . . 'panoramic' vision."

On the other hand, "The terrestrial carnivorous animals require for their sustenance and survival, smell and sight which will enable them to track their prey and pounce accurately upon it when they can concentrate the sight of both eyes on their victims at the distance from which they spring."

Then, referring to the primates, he goes on to say, "As the food of these arboreal mammals consists of fruits and insects . . . they require . . . a high degree of acuity of vision for small objects."

With this in mind, it is interesting to compare the ciliary muscle of these three classes of animals. The shape of the ciliary body varies largely with the amplitude of accommodation. In herbivora the ciliary body is spindle shaped and almost entirely confined to fibers corresponding to the meridional and radial groups (Henderson[7]). This is not to be interpreted as a rudimentary muscle, for the muscle fibers are generally numerous and well developed. In carnivora there is an apparent division of the muscle; circular fibers are present but make up only a small portion of the muscle mass. The ciliary body, as it is known in man, is not attained until the circular portion is further developed. It then becomes triangular.

In other words, those animals the requirements of which necessitate a constant adaptation for distance and which are known to have prac-

19. Collins, F. T.: Arboreal Life and the Evolution of the Human Eye, Philadelphia, Lea & Febiger, 1922.

tically no accommodation for near, appear to have a ciliary muscle composed exclusively of the noncircular portions. On the other hand, in those animals the requirements of which necessitate a ready change of focus for far and near, there appears the other muscle group, composed of circular fibers, in a degree proportionate to their respective accommodative amplitudes. If the ciliary muscle acts as a whole in the mechanism of accommodation for near, one would expect merely a hypertrophy of the already existing fibers rather than the appearance of an entirely new set. Or, conversely, one might expect a diminution of the ciliary muscle as a whole in those animals which have no near accommodation. The fact is that the ciliary muscle (meridional and radial fibers) may be well developed in the "nonaccommodating" herbivora.

I think it is fair to infer, therefore, that the meridional and radial muscles are associated phylogenetically with a focal adjustment for distance and the circular muscle with accommodation for near. A similar situation occurs in myopia and hyperopia in man. The myopic eye, being already in a state of adaptation for near, exerts relatively little demand on the circular fibers but exerts a normal or even greater demand on those fibers which are maintaining adaptation for distance. One therefore notes histologically a selective diminution of the circular fibers instead of a uniform ciliary atrophy.[20] The meridional and radial fibers are retained, and the ciliary body assumes somewhat the same shape seen in the lower animals (carnivora), which never did have a highly developed circular muscle. Conversely, in the hyperopic eye, which demands a greater accommodative amplitude, there develops a selective hypertrophy of circular fibers, not a generalized ciliary hypertrophy, and the shape becomes triangular in a manner unlike that in any of the lower animals.

d. Embryologic Factors.—In embryologic development one has an orderly recapitulation of the evolution of man. As accommodation for distance antedated accommodation for near by many phylogenetic eras, it is not surprising to find the noncircular portions of the ciliary muscle well developed in the human fetus before the appearance of any fibers of the circular group. The former appear well developed by the end of the fifth month, whereas circular fibers first appear in the sixth month, and the proportion of circular to noncircular fibers does not reach its final ratio until a varying time after birth.

20. In fact, the bulk of the ciliary muscle in myopia is often stated to be actually greater than in hyperopia.

FACTUAL CONSIDERATIONS

a. Neurologic Factors.—Clinical Observations: As I have previously suggested, if accommodation were purely a function of the parasympathetic system, lesions of the sympathetic system, whether paralytic or stimulatory, would have no influence on accommodation. That such is not the case is evident from the following case reports. As representative of paralytic lesions I have examined a group of patients who had been operated on by Dr. J. C. White with removal of portions of their sympathetic chain for Raynaud's disease, epilepsy, retinitis pigmentosa or some other condition. I have limited the subjects to those patients who had only one side operated on so that I might study the other for comparison. Since they all had Horner's syndrome postoperatively, I know I was dealing with a lesion of the sympathetic system, but it will be noted that Horner's syndrome was never complete. This is due to the fact that sympathectomy was done on only the lower cervical portion of the sympathetic system, and a complete Horner's syndrome is produced only after obliteration of the superior cervical ganglion or section of the posterior ganglionic fibers. Thus in the following cases it is only a sympathetic paresis and not a complete paralysis which I examined. I was not able to obtain for measurement a case in which sympathectomy was done on the superior cervical portion of the sympathetic system but I have every reason to expect that the findings in such a case would be even more marked.

CASE 1.—Agnes McG., aged 25, had a condition that had been diagnosed as Raynaud's disease. Three years previously the patient had sympathectomy done on the upper dorsal and the lower cervical portion of the sympathetic system on the right. A partial Horner's syndrome developed. The right pupil measured 2.5 mm. in diameter, and the left 5 mm. Refraction showed vision of 6/6 for the right eye with a + 0.25 D. sph. $\bigcirc$ — 0.37 D. cyl., ax. 180 and the same vision for the left eye with a + 0.50 D. sph. $\bigcirc$ — 0.37 D. cyl., ax. 180.

Accommodation (each figure represents the approximate mean of from ten to twenty measurements) was as follows: 9 D. for the right eye and 8.1 D. for the left eye, with direct measurement.

It was found, however, that the variation between successive observations was minimized by measuring the punctum proximum with a — 3 D. lens over each eye and allowing for this in the total accommodative power. I have called this the indirect measurement. With this measurement accommodation was as follows: from 10 to 10.5 D. for the right eye and from 8.5 to 9 D. for the left eye. In these measurements a pinhole of 2 mm. was used to eliminate any effect from the miosis alone.

When a — 5.50 D lens was placed before each eye and the eyes were examined separately, the patient observed that she could immediately overcome the lens with the right eye, but she experienced considerable difficulty in overcoming it with the left eye and succeeded in doing so only after an interval considerably longer than it took with the right eye. This significant observation was checked a number of times.

Believing that I should obtain a change in accommodation similar to the paradoxical pupillary reaction, I gave this patient 0.5 cc. of epinephrine hydrochloride subcutaneously, but obtained neither a pupillary nor an accommodative change.

CASE 2.—Daniel L., aged 19, had a condition that had been diagnosed as retinitis pigmentosa. Preoperative refraction showed vision of the right eye to be 6/12 with a —0.50 D. cyl., ax. 180 and that of the left eye to be the same with a +0.25 D. sph. ⊃ —0.50 D. cyl., ax. 180. The preoperative accommodation of each eye was 6.5 D. (mean value). The important point is that vision was the same for distance and for near, as far as the patient could judge

The patient had sympathectomy done on the upper dorsal and the lower cervical portion of the sympathetic system on the left. Horner's syndrome developed on the left. The pupil of the right eye measured 4 mm. in diameter, and that of the left eye measured 2.5 mm.; there were relative ptosis of 2 mm. and relative enophthalmos of 1 mm.

Postoperative refraction showed vision of the right eye to be 6/12 with a —0.50 D. cyl., ax. 180 and that of the left eye to be the same with a +0.75 ⊃ D. sph. —0.75 D. cyl., ax. 180. The postoperative accommodation of the right eye was 7.0 D., and that of the left eye was 8.5 D.

When the patient was asked if there was any difference between his eyes preoperatively and postoperatively, he observed that postoperatively it was relatively difficult for him to maintain clear vision for distance with the left eye, although a distant object seemed clear when he first looked at it. This was not true for reading distance. The apparent instability for distance was more marked four days postoperatively than when the patient was reexamined three weeks later, although it was still definite then.

CASE 3.—William G., aged 16, had a condition that had been diagnosed as epilepsy. Vision was noted six months preoperatively to have been 20/15 for each eye, and on his own account the patient stated that his vision had been equal in the two eyes.

Sympathectomy was done on the upper dorsal and the lower cervical portion of the sympathetic system on the left. A true Horner's syndrome developed. The patient was examined four days postoperatively.

Vision of the right eye was 6/5 without a glass. Vision of the left eye was 6/9 without a glass, but improved to 6/5 with a —0.75 D. sph.

Accommodation of the right eye was 12 D., and that of the left eye was 14.5 D. A 2 mm. diaphragm was used to exclude the effect of the miosis.

Subsequently the patient had sympathectomy done on the lower cervical portion of the sympathetic system on the left, and with the development of Horner's syndrome bilaterally accommodation was found to be equal in the two eyes.

CASE 4.—Elizabeth H., aged 46, had a condition that had been diagnosed as Horner's syndrome (on the right). Three weeks previously the patient had had a "cold," with an abscess of her right middle ear, and at that time a tender lump was noted on the right side of her neck. She had not noticed anisocoria until a few days previous to examination. The examination showed slight ptosis and enophthalmos on the right, with a pupillary measurement of 2.5 mm. for the right eye and 3.5 mm. for the left.

Refraction showed vision of the right eye to be 6/5 with a —0.50 D. cyl., ax. 180 and that of the left eye to be the same with a —0.50 D. sph. ⊃ —0.50 D. cyl., ax. 180. Accommodation of the right eye was from 3.5 to 4 D., and that of the left eye was 3 D.

With the right eye the patient overcame a —2.00 D. sph. readily over the aforementioned correction, and vision for reading was 6/6, but with the left eye she could overcome only a —1.50 D. sph. When a —2.00 D. sph. was placed before each eye, vision of the left eye was 6/12 at best and showed an indisputable difference when compared with that of the right eye.

CASE 5.—Dorothy R., aged 20, when first examined had Horner's syndrome on the right, which was said to have been present since birth. She also had depigmentation of the right iris.

Refraction showed vision of the right eye to be 6/6 with a + 1.12 D. sph. ⌒ — 0.25 D. cyl., ax. 120 and that of the left eye to be the same with a + 1.00 D. sph. ⌒ — 0.25 D. cyl., ax. 180.

Accommodation, as measured with a 2 mm. diaphragm, was as follows: With the right eye the patient could overcome a — 10.5 D. sph. and had vision for reading of 6/9, although it was not stable. With the left eye she could overcome a —9.0 D. sph., and vision for reading was 6/9, but she could not overcome an additional — 0.50 D. sph. even momentarily.

As the patient was a nurse and a fairly good observer, she was asked to note the difference between her two eyes while wearing minus lenses of different strengths. She observed that her ability to focus on a small letter (she used the 6/9 line) was less stable with her right eye than with her left eye. With the right eye the letters alternately faded and cleared, and she became fatigued more readily. This instability of focus with the right eye was present through the entire range of accommodation but was maximal when the eye was adapted for distance without any lens and at a minimum when the patient was using 5 D. of accommodation. She stated that she had been conscious of this instability of her right eye for years.

More significant still was the observation suggested by Dr. Verhoeff. When the patient was asked to fix with alternate eyes first on a near point and then at a distant one, she observed that it took a somewhat longer time for the distant object to come into focus with the right eye than with the left. When vertical diplopia was induced by a 5 prism diopter lens before one eye and the patient asked to look first at a near point and then at a distant one, she observed regularly that the distant image represented by the left eye came into focus more quickly and was maintained more efficiently than the image of the other eye.

On the other hand, when the patient was asked to note the difference in rate of accommodative change on looking from a distant point to a near object, she observed no difference either when she was using alternate eyes or when she observed the two images simultaneously with the vertical prism.

I was fortunate in obtaining the case history of one patient who presented the opposite condition, that induced by stimulation of the sympathetic chain. For this I am indebted to the observations of Dr. Verhoeff, who examined the patient a number of years ago. As evidence of the stimulation of the sympathetic chain there is the pupillary dilatation, in contrast to the miosis of Horner's syndrome.

CASE 6.—On Oct. 26, 1908, the patient was refracted and given the following prescription: a + 2.25 D. sph. ⌒ — 1.00 D. cyl., ax. 95 for the right eye (with which vision was 6/5) and a + 1.00 D. sph. ⌒ — 1.50 D. cyl., ax. 92 for the left eye (with which vision was 6/5).

On November 25, 1908, the patient returned. According to the record, the glasses were satisfactory until three or four days before, when vision of the right

eye was slightly blurred, especially on reading. During this time the patient had a slight cold, which had not developed into an actual cold but which was accompanied by chilly sensations. At the same time a tender spot was discovered on the right side, corresponding to the area of the superior cervical ganglion. The pupil of the right eye was slightly larger than that of the left eye. Each reacted to light and in accommodation, and the skin reflex was elicited ipsilaterally. Accommodation was slightly less in the right eye. The patient later accepted and was given a + 0.50 D. sph. in addition to the former correction. When reading through a pencil he required a + 0.50 D. sph. to make the letters appear equally distinct. The patient was examined later. The tender spot in the neck had disappeared, as had also the sense of blurring and the difference in accommodation, and he no longer tolerated the additional plus lens which had been given him.

It seems from the foregoing history that removal of portions of the sympathetic system results in a greater accommodative amplitude, while stimulation of the sympathetic system diminishes accommodative amplitude. In other words, stimulation of the sympathetic system tends to adapt the eye for distance and oppose the accommodation for near. These changes are not marked and might be overlooked in a routine examination, but they have been consistently present in the patients whom we have examined. It should be noted that most of the cases were those of recent postoperative Horner's syndrome, the effects of which may be lessened in the course of time with the regeneration of the preganglionic tracts. The fact that the lesions of the sympathetic chain in the neck have not resulted in more profound changes in the accommodation is in all probability due to the inherent autonomy of this function, like that of other functions under the influence of the autonomic nervous system. The nervous impulses vitally influence a function, but when deprived of the more proximal nervous connections the function can persist as an independent but less well ordered mechanism. Lesions of only one portion of the autonomic system, such as the sympathetic system, will impair the activity of this portion only. A paralytic lesion will render accommodation for distance less stable, while offering less resistance to accommodation for near. Stimulation has the opposite effect.

Experimental Observations: Before discussing the following experimental observations it may be well to recall briefly the structure of the autonomic system in relation to the eye.

The higher centers of the autonomic system are still disputable. But current belief would favor a center for sympathetic fibers in the hypothalamus (Karplus and Kreidl;[21] Beattie, Brow and Long;[22] Ran-

21. Karplus, J. P., and Kreidl, A.: Gehirn und Sympathicus: Ein Sympathicuszentrum im Zwischenhirn, Arch. f. d. ges. Physiol. **135**:401, 1910.

22. Beattie, J.; Brow, G. R., and Long, C. N. H.: Physiological and Anatomical Evidence for the Existence of Nerve Tracts Connecting Hypothalamus with Spinal Sympathetic Centres, Proc. Roy. Soc., London, s.B **106**:253, 1930.

son, Kabat and Magoun [23]). A similar center for parasympathetic fibers for the eye exists apparently in the midbrain below the aqueduct of Sylvius and perhaps in the hypothalamus as well. These two centers are fairly closely approximated, but the paths by which their impulses reach the eye are widely divergent.

The sympathetic fibers pass down the spinal cord to the inferior ciliospinal center of Budge (if this can be called a center), which is in the region of the lower two cervical and upper four thoracic segments. The sympathetic roots (white rami) leave the spinal cord in the region of the first three thoracic trunks, pass for the most part through the inferior cervical ganglion (or stellate ganglion, as it is called if fused with the first thoracic) and through the midcervical ganglion to reach the superior cervical ganglion. Here efferent neurons which have not terminated in the lower cervical ganglions undergo a synapse. It is to be noted that for each preganglionic neuron there are a varying number of postganglionic neurons. Thence the postganglionic fibers ascend to the base of the brain along the internal carotid artery. Here they form two interlacing plexuses called, respectively, the carotid plexus and the cavernous plexus. Thence sympathetic fibers enter the orbit with the blood vessels, with each of the motor nerves and particularly with the ophthalmic division of the trigeminal nerve. The latter innervates the intra-ocular structures by way of its nasociliary branch. The nasociliary nerve, which (it should be noted) has avoided the ciliary ganglion, gives off two long ciliary nerves. These are essentially sympathetic in nature.[24] These long ciliary nerves penetrate the sclera diagonally round the point of attachment of the optic nerve and pass forward in the perichoroidal space to take part in the elaborate plexus over the ciliary muscle. Thence terminal sympathetic fibers are contributed to the dilator muscle of the iris and, in all probability, to the ciliary muscle.

The parasympathetic fibers carrying impulses for near accommodation and pupilloconstriction do not have such a circuitous course. Coming from a center located in the midbrain, the impulses of the parasympathetic fibers are escorted directly into the orbit via the third nerve. They leave this nerve trunk after coming through the superior orbital fissure and enter the ciliary ganglion, where they pass through a synapse. The ciliary ganglion, except for a few sympathetic fibers, is a ganglion of the parasympathetic nervous system and, as was stated by Kuntz [25] in

23. Ranson, S. W.; Kabat, H., and Magoun, H. W.: Autonomic Responses to Electric Stimulation of Hypothalamus, Preoptic Region and Tectum, Arch. Neurol. & Psychiat. **33**:467 (March) 1935.

24. It is generally believed that a few sympathetic fibers from the carotid plexus are sent through the ciliary ganglion, but it is unlikely that they have a synapse here.

25. Kuntz, A.: The Autonomic Nervous System, Philadelphia, Lea & Febiger, 1929, pp. 317-323.

1929, constitutes a relay for peripheral functions. Its function is comparable to that which the superior cervical ganglion serves for impulses of the sympathetic fibers. After leaving the ciliary ganglion, the postganglionic parasympathetic fibers enter the eye through the short ciliary nerves. These, varying in number from twelve to twenty, form a cone about the optic nerve and proceed in the perichoroidal space to the aforementioned ciliary plexus. From the ciliary plexus are given off fibers to the iris and to the ciliary muscle.

It is apparent that after the sympathetic and parasympathetic fibers enter the eye an anatomic separation is practically impossible. As for the orbit, the only places where the paths are sufficiently divergent to allow separate excitation are, first, the long ciliary nerves or the nasociliary nerve from which they are derived and, second, the third nerve proximal to the ciliary ganglion. I exclude the significance of stimulating the ciliary ganglion itself because of the likelihood (Botar [26]) that it contains sympathetic as well as parasympathetic fibers. As for the neck excitatory or paralytic lesions here produce pure sympathetic effects as far as the eye is concerned. No parasympathetic fibers to the eye pass through the region of the neck. On the other hand, the only satisfactory place that a pure parasympathetic effect might be obtained with certainty would be in the intracranial portion of the third nerve.

With this anatomic discussion as a prelude, it is interesting to analyze certain pertinent experiments of others and a few of my own.

Most frequently mentioned is the classic work of Hensen and Voelckers,[27] published in 1868, which is said to prove that the ciliary muscle acts as a whole in accommodation. These authors observed changes in the ciliary body on stimulating the short ciliary nerves and the ciliary ganglion. The phenomenon of near accommodation was, of course, produced, as these tracts are predominately parasympathetic. The methods of these authors were necessarily gross, as retinoscopy was not known at that time. They inserted needles at right angles to and through the sclera in the regions overlying the ciliary body, the equator and the posterior pole. On stimulation of either the short ciliary nerves or the ciliary ganglion, there was no movement of the needles in the ciliary muscle or at the posterior pole, while the needle in the equator rotated in a direction indicating a forward displacement of the choroid. This displacement of the choroid was also observed directly by excising a window in the sclera and noting its gross movement. That these movements were accompanied by near accommodation was proved by putting

26. Botar, J.: Sur les particularités structurales des nerfs végétatifs de l'oeil, Bull. Assoc. anat., 1935, p. 15.

27. Hensen, V., and Voelckers, C.: Experimentaluntersuchung über den Mechanismus der Accommodation, Kiel, Schwârz, 1868.

a needle on the anterior surface of the lens and one on the posterior surface and noting their displacement on stimulation of the short ciliary nerves and the ciliary ganglion. The anterior surface was noted to move forward and the posterior surface backward.

That these observations made almost seventy years ago were accurate, there can be little doubt. But the conclusions which have been drawn from them are questionable. In the first place, the fact that one should get a forward displacement of the choroid in near accommodation does not prove that this displacement is due to the participation of the entire ciliary muscle. It is conceivable that contraction of only the circular fibers, which, it will be remembered, are situated at the anterior inner portion of the ciliary body, might alone exert a traction on the choroid in much the same way that the sphincter iridis muscle puts traction on the remote root of the iris. Furthermore, I might call attention to the probably dual nature of the short ciliary nerves and the ciliary ganglion. Though these are composed chiefly of parasympathetic elements, sympathetic fibers also are believed to be present, as was previously noted. Stimulation of one without the other would, of course, be impossible, although the parasympathetic fibers which predominate in these structures would preponderate in the functional manifestation of accommodation. I therefore go so far as to say that even though Brücke's muscle had been shown to contract on stimulation of these nerve fibers, one could not conclude that this was the case in the ordinary phenomena of near accommodation, nor could one conclude that such a contraction is the property of either branch of the autonomic system. On somewhat different grounds Barret [28] was similarly skeptical about Hensen and Voelckers' conclusions and stated, "They [the experiments] only prove that some movement took place within the eye on stimulation." Whether that movement was the selective action of Muller's muscle or of Brücke's muscle, separately or together, and, if the latter, what the significance of it was, is questionable.

By stimulation of the long ciliary nerves, however, or by stimulation of the cervical sympathetic chain in the neck, one would expect a pure sympathetic reflex. Both these structures have been stimulated. Jessop [29] in 1886 stimulated the long ciliary nerves of dogs and cats and observed the images of Purkinje and Sanson. The animals had previously had iridectomy. After rendering the animals somewhat myopic by pilocarpine he observed on stimulation a change in the direction of hyperopia. Morat and Doyon [6] in 1891 obtained similar results by stimulating the cervical portion of the sympathetic chain. By observing the size of the

28. Barret, J. W.: Do Mammals Accommodate? Ophth. Rev. **17**:255, 1898.

29. Jessop, M.: On the Anatomy, Histology and Psysiology of the Intraocular Muscles of Mammals, Proc. Roy. Soc., London **40**:478, 1886; Physiology of the Intraocular Muscles, Ber. period. internat. Ophth.-Cong. **7**:188, 1888.

images of Purkinje and Sanson, they noted on stimulation of the sympathetic chain a flattening of the lens. These measurements, made by the phakoscopic method, required a high degree of accuracy and were necessarily difficult to perform. Hess and Heine [30] in 1898, using the skiascopic method in a dog, showed in effect the same thing. They stimulated the cervical portion of the sympathetic chain and found an increase in hyperopia of from 1.0 to 1.5 D. Langley and Anderson [15] while studying movements of the iris made some incidental observations on accommodation. They used the phacoscopic method and were unable to observe any change in the images on stimulating the sympathetic chain. It is to be noted, however, that their observations were made largely on morphinized dogs. And morphine stimulates the centers for near accommodation. That stimulation of the sympathetic chain does not have the usual response in the eye of a morphinized animal is apparent from their own observation expressed later in the same article; they stated that under the influence of morphine the pupil dilates only slightly and then irregularly when the sympathetic chain is stimulated. Römer and Dufour [31] in 1902 were unable to produce a deflection of the needle resting on the anterior surface of the lens or to demonstrate a movement of a needle stuck in the ciliary muscle on stimulating the cervical portion of the sympathetic chain. Hess and Heine had also been unable to demonstrate a deflection of a needle thrust into the ciliary muscle on stimulation of the sympathetic chain, although the animal's hyperopia increased, as shown by skiascopy. Trautvetter [32] in 1866 also reported failure to get a response to stimulation of the sympathetic chain, but he apparently used dead animals only.

In other words, definite evidence that stimulation of the sympathetic chain induces a relative flattening of the lens has been demonstrated by several investigators. Most of those who have failed to demonstrate it have also failed to produce an adequate stimulation of the sympathetic chain that would at least cause the pupil to dilate well.

The amazing thing to me is not that some have been unsuccessful in demonstrating the sympathetic flattening of the lens but that those who have done so should not have realized its significance. Thus, Jessop [29] assumed that the effects must be due to a "relaxation" of the ciliary muscle. Morat and Doyon,[6] believing the same thing, suggested that the influence of the sympathetic nervous system was merely inhibitory.

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30. Hess, C., and Heine, L.: Arbeiten aus dem Gebiete der Accommodationslehre, Arch. f. Ophth. **46**:243, 1898.

31. Römer, P., and Dufour, O.: Experimentelle und kritische Untersuchungen zur Frage nach dem Einfluss des Nervus sympathicus auf den Accommodationsvorgang, Arch. f. Ophth. **54**:491, 1902.

32. Trautvetter, D.: Ueber den Nerv. der Accommodation, Arch. f. Ophth. (pt. 1) **12**:95, 1866.

That relaxation or inhibition is not the primary method of focusing for distance is apparent from direct observation of the capsule of the lens after discission and absorption of the cortical material. The tensile force exerted on the capsule is maximal in the so-called unaccommodated or "relaxed" state. When a person with the eye in such a state gazes at a distant object the capsule becomes relatively taut, and, conversely, it becomes folded on itself when the person attempts to accommodate for near (Graves,[33] 1926). It has been suggested that the elastic tissue inherent in the ciliary body keeps the lens in the flattened state while allowing stretching when the lens becomes accommodated for near. There are several points against this. In the first place, elastic tissue is not particularly abundant in the ciliary body; there is a moderate amount

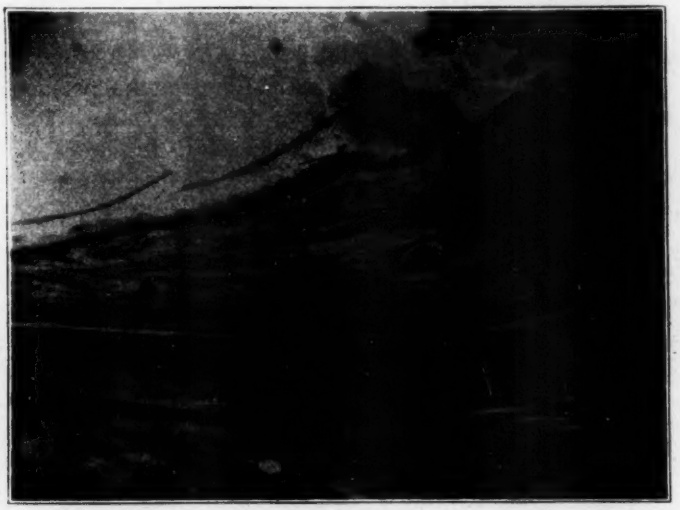

Fig. 1.—Photograph of a normal ciliary body stained with Verhoeff's elastic tissue stain, showing the paucity of elastic tissue in the ciliary body. There are practically no elastic fibers in the ciliary body except about the blood vessels, and there is a minute amount in the subepithelial tissue. As the ciliary body merges posteriorly with the more vascular choroid the elastic fibers become only slightly more numerous. In the region of the pectinate ligament there is an increase in the elastic tissue, but we are unable to agree with Fincham [2] that any one bundle would flatten the lens. The overlying sclera is richly supplied with elastic fibers and stands out by contrast in this photograph. The relative absence of elastic tissue in the ciliary body makes it necessary to postulate some force other than an elastic one to account for the phenomena of distance accommodation.

in the region of the pectinate ligament and in the sclera, but hardly any in the ciliary body itself (figure). Furthermore, a pure relationship

33. Graves, B.: Changes of Tension of the Lens Capsules During Accommodation and Under the Influence of Various Drugs, Brit. M. J. 1:4, 1926.

between muscles and elastic tissue, in which the two components are functionally antagonistic, would be an exception to the usual architecture of the autonomic system. Of interest also is the fact that a monkey or a human being becomes accommodated for near immediately after death, that is, as soon as the sympatheticotonic state of asphyxia is passed (this is a personal observation). I consider this further evidence for the muscular control of accommodation for distance as opposed to any inherent elasticity of the ciliary body. It is my opinion, therefore, that neither accommodation for near nor accommodation for distance can be appropriately called relaxed but that both manifestations are under the dynamic control of some portion of the ciliary muscle.

Hess and Heine,[30] after showing effectively that stimulation of the sympathetic chain caused an increase in hyperopia up to 1.5 D., suggested that with the accompanying hyperopia there was a dominance of the more peripheral lenticular refraction over the less hyperopic central portions. That this explanation was not justified is shown by my analogous experiment on a monkey and a dog, in which essentially the same procedure was used but the pupillary size was controlled by a 5 mm. aperture. I refracted the animals and then placed a plus glass before the eye, which just reversed the shadow. On stimulation of the cervical portion of the sympathetic chain there was a change in the direction of hyperopia of approximately 1 D. in the monkey and of from 0.5 to 0.75 D. in the dog. The artificial pupil did not change the findings appreciably.

A different type of experiment but one perhaps bordering on the same problem was reported by Pearcy and Allen[34] in 1927. These authors placed a balloon in the stomach and colon of their human subjects, and as the internal pressure was increased they noted a reduction of accommodation of from 2 to 5 D. Kuntz[25] in commenting on their work recalled that any somatic stimulation (e. g., pain) gives a sympathetic syndrome, as is evident in the eye from the pupillary dilatation.

In view of the foregoing clinical and experimental observations there would seem sufficient evidence to indicate that the sympathetic system had a considerable effect on the act of accommodation. Stimulation of the sympathetic chain favors a condition of adaptation for distance and antagonizes adaptation for near, while a lesion of the sympathetic system tends to impede adaptation for distance, while enhancing adaptation for near.

One may legitimately ask why adaptation for distance is at all possible when the afferent sympathetic fibers are cut and why the

34. Pearcy, J. F., and Allen, T. D.: Studies on the Visceral Nervous System: Reflexes from the Gastrointestinal Tract to the Eye, Am. J. Physiol. **82**:56, 1927.

reduction in accommodative amplitude is only from 1 to 2 D. The fact is that these sympathetic functions are capable of a great deal of autonomy, especially when the superior cervical ganglion and its postganglionic fibers are still intact. Here, again, the ciliary muscle is much like the pupil, for not only are the dilator fibers still functioning many years after cervical sympathectomy, but they react even more energetically to epinephrine than does the normal pupil ("paradoxical pupillary reaction" in the sense that Duke-Elder defined it). This autonomy, as suggested by Byrne,[11] may be a reversion to a more primitive form of stimulation, that of control by hormones, or, as Cannon and Rosenbleuth[35] suggested, it may be due to increased permeability of the sympathectomized structures. In any case one cannot expect an atrophy of the muscles supplied by the sympathetic system if they retain such a potential reaction to the proper stimulus. I was interested in examining the two eyes of a monkey which had had a unilateral sympathectomy several months previously. I was unable to demonstrate any difference in the ciliary muscles of the two eyes.

b. Pharmacologic Factors.—The autonomic nervous system is inextricably bound up with the hormonal system. In all probability the nervous impulse results in muscular contraction only through the generation of its specific hormone. It is stated that only one in twenty of these individual muscle fibers receives a nerve ending. The others must be stimulated through a chemical or hormonal medium. The hormone for the sympathetic system is epinephrine, when produced by the adrenal glands, or its closely allied compound sympathin, when generated at the nerve ending. The injection of epinephrine hydrochloride produces in effect, then, a state of sympathetic stimulation similar to that produced by putting an electrode on the sympathetic fibers.

Accordingly, another investigator and I were given subconjunctival injections of epinephrine hydrochloride in one eye and subsequently made careful observations on the relative accommodative power of each eye, the eye into which injection had been made and the normal eye.

EXPERIMENT 1.—(D. G. C.).—Accommodation was measured a number of times before the injection; a mean value of 8 D. was obtained for each eye. Incidentally, the variability in establishing a mean was minimized by measuring accommodation with a — 3 D. lens over each eye and then finding the punctum proximum for Jaeger's test type no. 2. The 3 D. was then added to the near point measured in diopters. In this way the punctum proximum was removed to a point which allowed a greater distance per dioptric unit. All the observations were made with 2 mm. diaphragms to prevent erroneous conclusions resulting from inequality of the pupils. Two minims (0.12 cc.) of a solution of epinephrine hydrochloride was injected into the lower cul-de-sac of the left eye. In ten minutes, accommodation of the right eye was 7 D. (mean value), while accommodation of the left eye was reduced to between from 5.5 to 5.75 D. Practically the same inequality was

35. Cannon, W. B., and Rosenbleuth, A.: The Sensitization of a Sympathetic Ganglion by Preganglionic Denervation, Am. J. Physiol. **116**:408, 1936.

present in twenty minutes. In forty-five minutes accommodation was 8 D. in each eye, although the pupil was still dilated, showing again that the pupillary inequality was not responsible for the apparent difference.

The difference in the two eyes was also strikingly shown in the following manner. A —5 D. lens was placed before each eye, and the eyes were then alternately covered. With the right eye the —5 D. lens could be readily overcome; vision of 6/6 was obtained within a fraction of a second and maintained indefinitely. With the left eye, however, vision of 6/6 could be obtained with some difficulty and only after an interval considerably longer than with the other eye. Nor could it be maintained as stably as with the other eye, giving the impression of clearing momentarily and then fading.

EXPERIMENT 2.—(M. R. S.).—The method of examination and the results were practically identical with those in the previous experiment. The mean accommodative amplitude with the pupillary diaphragm was 8 D. for each eye. Two minims of epinephrine hydrochloride was injected subconjunctivally into the left eye. Twenty minutes later the accommodative amplitude of the right eye was between 8 and 8.25 D. and the amplitude of the left eye was 6.5 D. With a —5 D. lens over each eye, vision of 6/6 was obtained readily and maintained with the right eye; it was obtained with greater difficulty and maintained less well with the left eye.

The action of the sympathomimetic drug epinephrine has, therefore, an effect similar to that of sympathetic stimulation in opposing accommodation for near. With the small doses used, however, we were unable to demonstrate an increase in the hyperopia. Similarly, Heath [36] in a recent communication reported a partial loss of near accommodation after instillation of the sympathomimetic drug neosynephrin hydrochloride.

Basil Graves [33] reported an interesting case, which has already been referred to, showing the effect on the capsule of the lens of cocaine, which has a sensitizing effect on the sympathetic system. The patient had apparently had a spontaneous absorption of the entire cortex and nucleus of the lens after a perforating injury. All that was left of the lens was the anterior and the posterior capsule, with a small hole in the former; the two capsular membranes were separated by aqueous. Graves observed with the slit lamp that the membranes were relatively taut when the patient focused for distance and became folded on themselves when the patient attempted to look at something near at hand. When cocaine was instilled the capsules became quite taut and ultimately showed no alteration when the patient focused for near. That this striking effect was not due to the accompanying mydriasis was shown by the fact that the buckling of the capsules on near accommodation returned before there was any diminution in the size of the pupil. If, then, as is generally believed, cocaine sensitizes the sympathetic system,

36. Heath, P.: Neosynephrin Hydrochloride: Some Uses and Effects in Ophthalmology, Arch. Ophth. **16**:839 (Nov.) 1936.

one has a unique demonstration of this effect directly on the capsule of the lens. Graves did not, so far as I know, use epinephrine.

I have already referred to the work of Poos,[8] who showed much more striking effects from epinephrine than I did. He used instillations of epinephrine hydrochloride in patients who had had one eye sensitized by a previous unilateral lesion of the sympathetic system. This enhances the response of a structure of the sympathetic system to epinephrine. In contrast to the reduction in accommodative amplitude of 1.5 D. in our own eyes, Poos found in these sensitized eyes a reduction of 4 D. in persons having the same normal amplitude as our own.

It is interesting to review the effects of one other drug about which a good deal is known. That is atropine. Atropine is believed to "fix" or render inert the hormone acetylcholine which mediates the activity of the parasympathetic system. It thereby produces an effective paralysis of the parasympathetic system. Such being the case, the unopposed sympathetic system might be expected to have an effect greater than usual under atropinization, and this is what one finds. Under the influence of atropine apparent hyperopia increases anywhere from 0.5 to 1 D. In myopia, on the other hand, in which there is a selective deficiency in the circular muscle, one expects the sympathetic system to be acting more nearly at its maximum effectivity all the time. One would then expect in myopia relatively little refractive change under the influence of atropine. This, of course, is what one finds; the myopic eye appears to change little, if at all, under the influence of atropine.

CLINICAL SPECULATIONS

What bearing the aforementioned concept has on clinical problems is not the primary concern of this paper. But the applications are undoubtedly manifold, and it may be well to mention a few.

Hyperthyroidism, for instance, produces a syndrome similar to that resulting from generalized stimulation of the sympathetic chain, and the ocular signs of hyperthyroidism are like those produced by stimulating the sympathetic chain. There are widening of the palpebral fissure, dilatation of the pupil and, frequently, exophthalmos, as well as an abnormal sensitivity to epinephrine (Loewi's phenomenon). I have had occasion to measure the accommodation in a number of patients during the active stages of hyperthyroidism. The results were not striking, but approximately one quarter had an accommodative amplitude less than the normal minimum for their age, Duane's [37] chart being used as the standard. I do not attach too much significance to this, as the measurement of accommodation is notably variable in any group of persons. However, it was my impression that those who had a

37. Duane, A.: Studies in Monocular and Binocular Accommodation with Their Clinical Applications, Am. J. Ophth. **5**:865, 1922.

diminution in their accommodation and many of the others who showed a normal range did have poorly sustained accommodation and early fatigue, compared with a normal group.[38] It was much like the relative mydriasis which many patients with hyperthyroidism have; light causes immediate miosis but it is poorly sustained, and hippus becomes pronounced. In any case, asthenopia is a notably frequent symptom of hyperthyroidism, varying, as Davis [39] pointed out, from "moderate discomfort to inability to use the eyes at all for near work."

Grancher [40] reported an interesting case in this connection. His patient was a 37 year old man who could not read during his hyperthyroidism without the aid of a plus glass. No actual measurement of his accommodation was made, but when the hyperthyroidism improved he again discarded his reading glass.

Many of the anomalies of accommodation, such as ill sustained accommodation, transient weakness of accommodation associated with migraine, and excessive accommodation, may be due in part to functional disturbances of the autonomic nervous system. As Hudelo [9] pointed out, the alleged spasm of accommodation in asthenopes and other persons with visual disturbances is probably not a primary spasm at all but really a weakness of the muscles controlling distance adjustment. Conversely, an increased tone of muscles innervated by the sympathetic system may lead to inadequate near accommodation. A case illustrating such a functional disturbance was brought to my attention recently by one of the Harvard University medical examiners, who stated that such cases were common in his experience. A junior in the college came up for final semester examinations. On the morning of the first examination he went down for breakfast and noticed that the menu was blurred. In fact, anything which he tried to read became blurred. Somewhat alarmed, he reported to the Hygiene Building, where, aside from the blurring for near, the only findings were relatively dilated pupils, giving a startled appearance. The pupils, however, reacted promptly to light. The boy had been well up in his studies, and he was not thought to be a malingerer. He was, therefore, sent to the infirmary, and within a few hours, without any treatment, he was able to read without difficulty.

It may be significant that these anomalies occur in that large group of neuroses about which little is known but in which there are other evidences of disequilibrium of the autonomic system. This would not apply, of course, to the anomalies secondary to organic changes within

38. G. Rossi (Tempi di accommodazione in ipertiroidei, Arch. di ottal. **40**:483, 1933) reported that the time for near accommodation in patients with exophthalmic goiter is retarded by from two-tenths to three-tenths second.

39. Davis, W. T.: Ocular Symptoms of Thyroid Dysfunction, South. Med. & Surg. **95**:357, 1933.

40. Grancher: Goitre exophthalmique, Gaz. d. hôp. **53**:1060, 1880.

the eye, but it might apply to the anomalies which, usually transient in occurrence, come on, like other disturbances of the autonomic system, at times of worry or emotional distress.

SUBSEQUENT NOTE.—Since writing this paper I have come across a post-war study of Horner's syndrome in soldiers.[41] The authors reported that the near point was from 1 to 4 cm. nearer on the affected side than on the normal side. So far as I know, this is the first report of unequal accommodative amplitude in Horner's syndrome.

ABSTRACT OF DISCUSSION

DR. WILLIAM H. LUEDDE, St. Louis: Dr. Cogan found an increased capacity to accommodate, varying from 0.5 to 2.5 D., with marked contraction of the pupil on the same side, after surgical or other damage to the cervical portion of the sympathetic system in a series of five cases. Was there a control series? Was the refraction measured with the eye in complete cycloplegia? Does a 2 mm. diaphragm before the eye necessarily eliminate the effect of the inequality of the pupils?

Hess found that an apparent change of refraction of 1.5 D. or more can result solely from contraction of the pupil without any actual change in accommodation. He stated that a test with some instrument based on the principle of Scheiner is desirable to ascertain the certainty of an actual accommodative change. Were such corroborative tests made?

The history of Dr. Verhoeff's patient may be suggestive but certainly is not actual proof of innervation of the ciliary muscle by the sympathetic system. Consideration must be given to the possible influence on the accommodative capacity of the increased volume of blood in the ocular vessels after sympathectomy on the cervical portion of the sympathetic system due to paralysis or degeneration of the vasoconstrictor nerves.

The idea of an antagonistic set of muscles in the ciliary body like the dilator and sphincter fibers in the iris fails to materialize on close approach. The author has evidently overlooked Poos' statement that while he was able to produce direct contraction of isolated fibers of the dilator muscle of the iris by epinephrine, all attempts to secure similar results on preparations of muscle fiber from the ciliary body of various animals resulted in failure. That observation seems to cast much doubt on the claims for an active rôle of the sympathetic nerves in the ciliary musculature.

Stotler and Clark, working independently, reached identical results. Operative removal of the superior cervical sympathetic ganglion in the cat results in degeneration of the fibers supplying the dilator muscle of the iris but not of those supplying the sphincter muscle of the iris or any portion of the ciliary muscle. Operative removal of the ciliary ganglion in the cat results in complete degeneration of the fibers supplying the sphincter muscle or the iris and the ciliary muscle.

41. Cobb, Stanley, and Hunter, W. Scarlett: A Report of Eleven Cases of Cervical Sympathetic Nerve Injury, Causing the Oculopupillary Syndrome, Arch. Neurol. & Psychiat. 3:636-652 (June) 1920.

Shaklee, Christensen and Kaplan found evidence of an inhibitor of sympathetic impulses on terminal fibers of the sphincter muscle of the iris but none whatever in the ciliary musculature. These reports are in full accord with the conclusions of Cannon and Rosenblueth.

The pharmacologic effects resembling those caused by stimuli of the sympathetic nerves reported by the author after injections of epinephrine hydrochloride, the effect of neosynephrin hydrochloride observed by Parker Heath and the improved cycloplegia obtained by Beach when benzedrine was combined with homatropine or atropine are probably the result of chemical inhibition in the formation or direct destruction of the acetylcholine-like substance which carries the parasympathetic impulse from the myoneural junction into the muscle cell.

What supports the proposition that so-called distance accommodation is speedier because sympathin acts on smooth muscle fibers more quickly than acetylcholine? Cannon and Rosenblueth's report on the minimal effective amounts of sympathin and acetylcholine required to evoke detectable responses on reaching a smooth muscle cell is 0.00002 Gm. for acetylcholine and three times as much, 0.00006 Gm., for sympathin.

I doubt the validity of the suggestion that primates have the greatest range of accommodation.

Any explanation for the mechanism of accommodation which fails to envisage the fact that advance of the vitreous is an inevitable sequence to contraction of the ciliary muscle is inadequate.

I would protest against the possible implications of the term gross as applied by the author to the observations of Hensen and Voelckers, made in 1867, because their work antedated the use of retinoscopy.

Donders wrote a comprehensive treatise on the "Anomalies of Refraction" without the benefit of retinoscopy.

DR. ALFRED BIELSCHOWSKY, Hanover, N. H.: Ever since I found Sherrington's law of reciprocal innervation confirmed by my own experimental and clinical investigations of the motor apparatus of the eye I have been wondering why accommodation should be the only mechanism in which two antagonistic functions would be controlled by one single nerve. In 1900, when I had discussed at the meeting of the Heidelberg Ophthalmologische Gesellschaft physiologic and clinical observations which could be explained only by the assumption of an independent mechanism for divergence, Gullstrand told me that he did not agree with my argumentation based on Sherrington's law. He stated the belief that relaxation of convergence is sufficient to produce the change from the convergent, to the parallel, and even to the divergent, position of the eyes.

It is hard to realize how the rapid and yet delicately graduated adaptation of the eye to different distances could be brought about against the resistance of elastic tissues by one nerve controlling the contraction and relaxation of the ciliary muscle.

As long ago as 1856 A. von Graefe considered it improbable that the accommodative changes are controlled by a single nerve, and W. Henke in 1860, referring to the analogy between the structure of the iris and the ciliary muscle and their intimate functional relationship, called for a correspondingly analogous innervation of the muscles which produce both pupillary and accommodative changes. A double innervation of the ciliary muscle was conceivable in view of its structure.

Since stimulation neither of the long ciliary nerves nor of the superior cervical ganglion gave unequivocal results, Poos resorted to pharmacologic stimulation of the endings of the sympathetic and parasympathetic nerves in the iris and the ciliary body. The results he obtained in normal persons when compared with those of patients suffering from lesions of the cervical sympathetic chain supported the assumption of the double innervation of the ciliary body.

Dr. Cogan's interesting observations and experiments represent an important confirmation and enlargement of Poos' findings. Poos experimented with cocaine and different epinephrine preparations. Cocaine affects the sympathetic nerves supplying the smooth muscles of the eye and its adnexa. Apart from its stimulating influence on the sympathetic nerves, cocaine is supposed to have a paralyzing effect on the parasympathetic nerves supplying the iris and the ciliary body. But this assumption cannot be reconciled with the fact that if the sympathetic fibers are paralyzed cocaine produces neither dilatation of the pupil nor paresis of accommodation, a fact suggesting that the effect of cocaine on the pupil and accommodation of normal persons may be due to stimulation of the sympathetic nerves rather than to paresis of the parasympathetic nerves. Moreover, there is a striking difference between the paresis of accommodation caused by atropine or homatropine and the paresis caused by cocaine, epinephrine or ephedrine.

Slides are demonstrated, one of which shows a graph from Poos' article, while the others illustrate the results of experiments made in our clinic. The curves show that incomplete paresis of accommodation develops as rapidly as it disappears, whereas a curve illustrating the effect of atropine shows a much slower ascent but remains for a considerable length of time at the maximum height and then returns, rather slowly, to the normal level. The difference in behavior of the accommodation just discussed is similar to the behavior of the pupil according as to whether the dilator muscle is stimulated or the sphincter paralyzed. Thus it is conceivable that cocaine and epinephrine produce not paresis of the ciliary muscle but stimulation of that portion which is controlled by the sympathetic nerves, so that while the value of the range of accommodation remains the same its position moves off with reference to the eye. If that stimulation would produce flattening of the lens one would expect a decrease of refraction. To ascertain this might be difficult, if possible at all, without the antagonistic function of the ciliary muscle being eliminated. Considering that the maximum mydriasis caused by paralysis of the sphincter muscle can be increased by cocaine, one should try to ascertain whether a decrease of refraction could be obtained by cocaine and epinephrine in suitable cases. Such a decrease would have to be attributed to flattening of the lens as a result of stimulation of the portion of the ciliary muscle controlled by the sympathetic nerves. Preliminary experiments that we have made in our institute gave negative results. After the accommodation had been completely paralyzed by atropine no decrease of the refraction could be obtained by repeated instillations of a 10 per cent solution of cocaine.

Dr. Cogan's observations on patients with an incomplete Horner's syndrome who show an increase of accommodation strongly suggest innervation of a part of the ciliary muscle by the sympathetic system.

Dr. Cogan could not ascertain a decrease of refraction in his patients or obtain it with subconjunctival injections of epinephrine in normal persons. There was just a slight diminution of accommodation according to a record concerning Dr. Verhoeff's patient with stimulation of the sympathetic fibers on one side.

Thus one may state that not only theoretical considerations but experimental and clinical observations call for innervation of the ciliary muscle by both the sympathetic and the parasympathetic system to cooperate in distance accommodation as well as in near accommodation according to Sherrington's law. For the time being, however, there is no irrefutable evidence as to flattening of the lens brought about by an independent action of the portion of the ciliary muscle controlled by the sympathetic nerves.

Dr. S. Judd Beach, Portland, Maine: As clinicians, ophthalmologists are too prone to neglect accommodation. Dr. Luedde mentioned, in connection with this subject, work by McAdams and myself with benzedrine. As was reported at the meeting of the American Ophthalmological Society, by using benzedrine with cycloplegics one avoids the nuisance of multiple instillations and at the same time speeds up recovery. When in one hour one can make one instillation of atropine and one of benzedrine do the work of nine instillations of atropine over three days the temptation is to explain this synergism according to the theory just defended by the author. While one must take care not to be seduced by this hypothesis merely because it is convenient, every evidence in favor of it is of service, and I certainly hope that Dr. Cogan will continue his investigations.

Dr. D. G. Cogan, Boston: I fear that in the few minutes I have had to think them over and in the fewer minutes that I have to discuss them I shall not be able to do justice to Dr. Luedde's criticisms, but I should like to consider as many of his comments as time permits.

First to be considered is the matter of control. I do not know what I would use as a control series. If I used normal persons with normal accommodation on the two sides I do not know which of several thousands I would choose for comparison. I should say, however, that the accommodative characteristics peculiar to Horner's syndrome are not duplicated by any constant difference in accommodation that I have seen in a series of normal persons.

Dr. Luedde suggested using a cycloplegic. Cycloplegics such as atropine and homatropine paralyze exclusively the functions of the parasympathetic system and consequently have varying effects in normal and sympathectomized eyes. In the case of the pupil, for instance, atropine produces only semimydriasis on the side on which sympathectomy was performed, in contrast to the full mydriasis on the normal side. It is my contention that comparison of these atropinized ciliary bodies would be open to the same objections as comparison of these atropinized irides; the fact is that a cycloplegic does not necessarily reduce the normal and sympathectomized eyes to a state of comparable equality, and I have purposely avoided drawing any conclusion from the findings when the eye is in cycloplegia.

Dr. Luedde suggested that the circular fibers are merely continuations of the radial group. They certainly are continuous, but this is no objec-

tion to their separate functions, as is evident in the inseparable but antagonistic muscles of the gastro-intestinal tract, or in the case of the iris, where the dilator fibers are intimately connected with the sphincter muscle.

Dr. Luedde said that the effects of the drugs reported probably indicate inhibition of the parasympathetic fibers rather than primary stimulation of the sympathetic fibers. This is a subject one might profitably dwell on at length if time permitted. The whole concept of inhibition infers that there are elastic fibers antagonizing the action of the ciliary muscle. I was rather surprised when I examined the ciliary body histologically to find a remarkable paucity of elastic fibers in this structure. This is one of the major objections to the concept. Without going into it more deeply, let me say that evidence to corroborate inhibition as the entire *vis a tergo* in distance accommodation is lacking, and until such evidence is forthcoming I do not think that one can justifiably use it to explain the action of these drugs.

I should like to emphasize again the importance of using the impartial terms near accommodation and distance accommodation rather than accommodation and relaxation of accommodation. It is not a mere quibble, for the latter terms, particularly relaxation, imply a hypothesis which it is the purpose of this report to show is inadequate. The intention of this work is to evaluate the evidence bearing on "relaxation." If the concept is found wanting, so is the name which implies it.

Let me express my sincere gratitude for Dr. Luedde's comments. Controversy makes the subject all the more interesting. I know that both Dr. Luedde and I are fascinated by this interesting subject, the last of which, I am sure, has not yet been heard. I should also like to thank Dr. Bielschowsky, with whom, of course, I am in entire accord, and also Dr. Beach for his contribution to the pharmacology of the subject.

BILATERAL ATROPHY OF THE OPTIC NERVE IN PERIARTERITIS NODOSA

A MICROSCOPIC STUDY

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In 1929, we [1] described characteristic necrotizing lesions of the media and proliferation of the intima of the choroidal arteries in a case of periarteritis nodosa. Christeller [2] had mentioned arterial changes in the choroid, while Müller [3] had described characteristic changes in the retinal arterioles many years before. In the year our case was reported, other instances were described by Böck [4] and von Herrenschwand.[5] Böck's patient showed typical lesions in the extra-ocular muscles, and in the ciliary vessels at the entrance of the optic nerve and in their course through the sclera to the suprachoroidea. Several ocular muscles were paretic, and there was reduction in the pupillary reaction to light. The choroid was normal. In von Herrenschwand's case the lesion affected the long posterior ciliary arteries. The central retinal artery was diseased at its entrance into the optic nerve and in the region of the lamina cribrosa. The choroidal vessels were relatively uninvolved.

Helpern and Trubek [6] described lesions in the choroid in a case of gonococcic endocarditis accompanied with necrotizing arteritis which were almost identical with the choroidal lesions in our case (arteritis and proliferation of the intimal cells). This coincidence may be considered a substantiation of the view that the lesion of periarteritis nodosa is a symptom of a generalized infectious or toxic process for which no single organism can be held responsible. As far as the eye is concerned,

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From the Laboratories and Ophthalmological Division of the Mount Sinai Hospital.

1. Goldstein, I., and Wexler, D.: The Ocular Pathology of Periarteritis Nodosa, Arch. Ophth. **2**:288 (Sept.) 1929.

2. Christeller: Arch. f. Verdauungskr. **37**:249, 1926.

3. Müller, in Festschrift zur Feier des fünfzigjährigen Bestehens des Stadt-Krankenhauses zu Dresden-Friedrichstadt, Dresden, W. Baensch, 1899, p. 458.

4. Böck: Ztschr. f. Augenh. **69**:225, 1929.

5. von Herrenschwand: Klin. Monatsbl. f. Augenh. **83**:419, 1929.

6. Helpern, M., and Trubek, M.: Necrotizing Arteritis and Subacute Glomerulonephritis in Gonococcic Endocarditis, Arch. Path. **15**:35 (Jan.) 1933.

Friedenwald and Rones,[7] in discussing the relation between arterial lesions in the choroid in periarteritis nodosa and those seen in septicemia, found them to be closely related, in that periarterial involvement of one kind or another was the rule. As regards the presence of so-called albuminuric retinitis, which is often seen in periarteritis nodosa when complicated by hypertensive and renal disease, they found only sclerosis of the retinal arterioles to account for the retinitis in their case. This seemed to answer the question set forth by von Herrenschwand as to whether the retinitis might be due to inflammatory lesions of the arterioles. King[8] noted papilledema resulting in atrophy of the optic nerve in a case of periarteritis nodosa, in which diagnosis was made through biopsy. In the other eye there were recurrent iritis and glaucoma which necessitated enucleation. This eye revealed retinal arteritis and periarteritis, but the lesion was not typical of periarteritis nodosa.

In Böck's[9] second case there was disease in the scleral and choroidal vessels and in the greater arterial circle of the iris. In the same article in which this case was described another case was reported, which was most interesting. In a boy of 17 progressive total detachment of the retina developed in each eye during the course of a condition proved by histologic examination to be periarteritis nodosa. With improvement in the general condition each retina reattached spontaneously, although the blood pressure had risen to 220 systolic and 165 diastolic. Several months later, vision was 20/100 in each eye. There were secondary atrophy of the optic nerve and, most striking of all, strands of pigmented tissue crossing through the fundus in its subretinal layers (retinitis striata) and apparently binding the retina to the choroid. The macula was pigmented, and below it were groups of circumscribed small masses of pigment. The arteries and veins were narrow. Under the conditions, nephritis was considered in the etiology, but Böck considered this improbable, since the patient's condition had improved, the blood urea content was not excessively high, and there was not sufficient hypertension to account for the bilateral detachment; in fact, the systolic blood pressure was only 160 and rose considerably only after reattachment. Extensive subretinal exudation as a consequence of diffuse arterial disease in the choroid was held more likely to be responsible.

The case to be reported is similar in some respects to Böck's last case, in that there were bilateral consecutive atrophy of the optic nerve and considerable pigmentary disturbance in the fundus. In addition to atrophy of the optic nerve, microscopic examination revealed extensive disease of the short posterior ciliary and choroidal arteries in each eye.

7. Friedenwald, J. S., and Rones, B.: Ocular Lesions in Septicemia, Arch. Ophth. **5**:175 (Feb.) 1931.

8. King: Tr. Ophth. Soc. U. Kingdom **55**:246, 1935.

9. Böck: Ztschr. f. Augenh. **78**:28, 1932.

REPORT OF A CASE

History.—Mrs. C. D., aged 45, had asthmatic attacks sixteen months before admission to the hospital. During the last six months she had lost much weight and suffered extreme muscular weakness. Four months previously, biopsy of a specimen of muscle at another hospital showed periarteritis nodosa. Wrist drop developed on the left. Vision suddenly became poor in both eyes. Recently there had been dyspnea, cyanosis and general parasthesias.

Physical Examination.—The patient appeared cachectic and irrational. The veins of the neck were engorged. There were several millet-sized nodules of bony consistency on the forehead. There were small nodes in the neck and others in the axillae, epitrochlear regions and groins. There were congestive signs in the lungs. The heart was enlarged, and a pericardial friction rub was heard. The liver was slightly enlarged; the spleen was not palpable. There were signs of wrist drop on the left and paralysis of the median nerve in the left hand. The blood pressure was 115 systolic and 110 diastolic. The hemoglobin content was 83 per cent. The red cells of the blood numbered 4,430,000, and the white cells 19,250, of which 91 per cent were polymorphonuclear leukocytes. The urine showed a faint trace of albumin; its concentration power was up to 1.028. There were 75 mg. of urea per hundred cubic centimeters of blood, 75 mg. of sugar and 165 mg. of cholesterol. The carbon dioxide content was 25 volumes per cent; the uric acid content was 6 mg. per hundred cubic centimeters

Externally, the eyes were clear. The pupils were semidilated and barely reacted to a stimulus of strong light. Vision of each eye was reduced to perception of movements of the hand. The fundi were similar in appearance. In the right eye the disk was pale and had a greenish tinge. There was a sharp choroidal ring in the nasal portion, but the other margins were indistinct. The surface margins of the disk were obliterated by glial tissue, only a small part of the physiologic pit being left visible. The patient was a decided brunette; however, the regular pigmentary pattern of the choroid had disappeared throughout the fundus, leaving only a diffuse fine scattering of granules of pigment. There were no clumps of pigment. The retina appeared thin, but there were no white lines or streaks. The retinal arteries and veins were narrow. The diagnosis was consecutive atrophy of each optic nerve, and it was thought that the disturbance in the pigment might be accounted for by disease in the choroidal arteries, although there was no ophthalmoscopic evidence to substantiate this belief.

Course.—The patient remained irrational. Her condition became rapidly worse. There was a preterminal rise in the temperature to 104 F. Death occurred nine days after admission to the hospital.

Autopsy.—There was periarteritis nodosa involving the vessels of the kidneys, liver, mesentery, diaphragm, spleen, lungs and heart. There were also diffuse cortical scarring of the kidneys, subcapsular atrophy and congestion of the liver, subacute suppurative pericarditis, hypertrophy and dilatation of the right auricle and ventricle and myocardial fibrosis. There were general edema, ascites, bilateral hydrothorax, and bronchial pneumonia of all the lobes of the lung.

Both globes, together with 5 mm. of the optic nerve of each eye, were removed eight hours after death. Fixation was done in Bouin's solution, pyroxylin (celloidin) and paraffin, and staining, with hematoxylin and eosin.

Microscopic Examination of the Eyes: The vessels of the iris and ciliary body were normal. The central retinal artery and the retinal arterioles, as far as they could be traced, showed no change in either eye. Changes in structure and in the vessels were confined to the episclera, the choroid and the disk in each eye.

The choroid was of normal thickness. Most of the medium-sized arteries showd infiltration of the media with mononuclear and round cells. In some there was necrosis in addition to dense infiltration; others showed proliferation of the intima, the formation of fibrin and obliteration of the lumen (fig. 1 *A* and *B*).

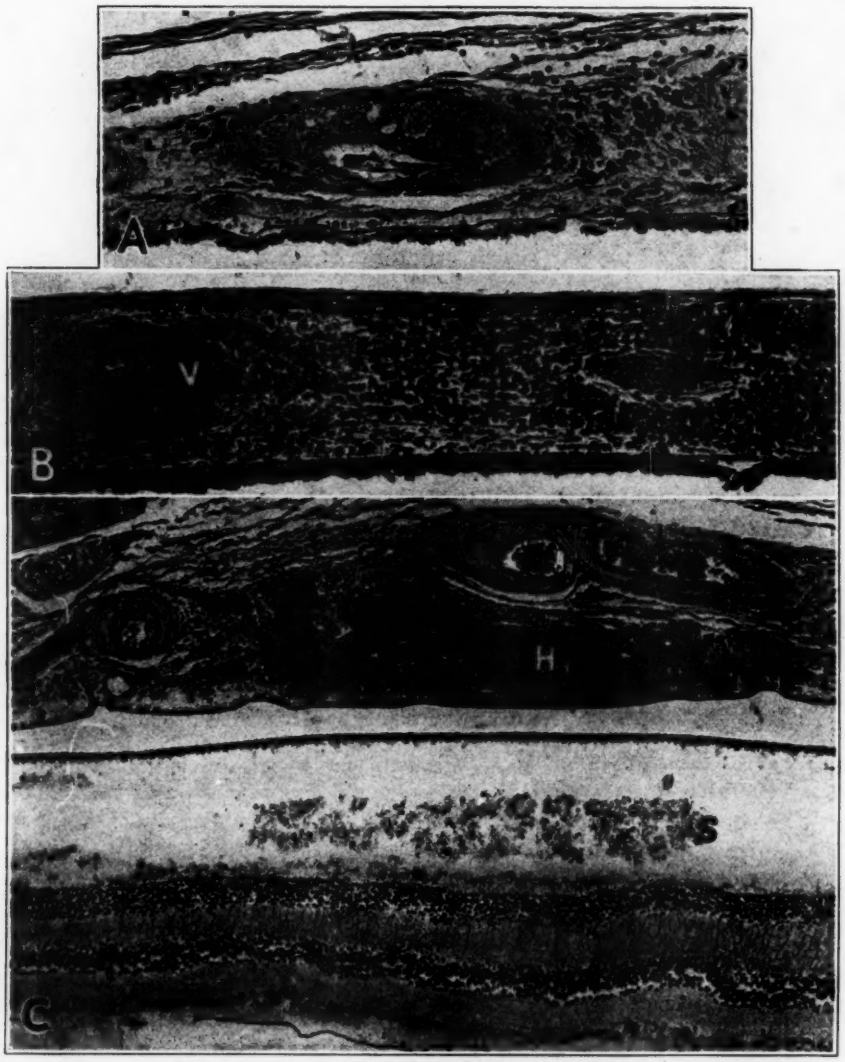

Fig. 1.—*A*, section of a medium-sized artery of the choroid, showing necrosis of the media and adventitial and perivascular infiltration. *B*, thickly infiltrated choroid; *V* indicates a necrotic infiltrated artery. The choroid is thickly infiltrated with mononuclear and plasma cells. *C*, large choroidal hemorrhage, *H*, resulting from thrombosis of a small choroidal artery; *S*, indicates subretinal exudate. The layer of rods and cones is disintegrated.

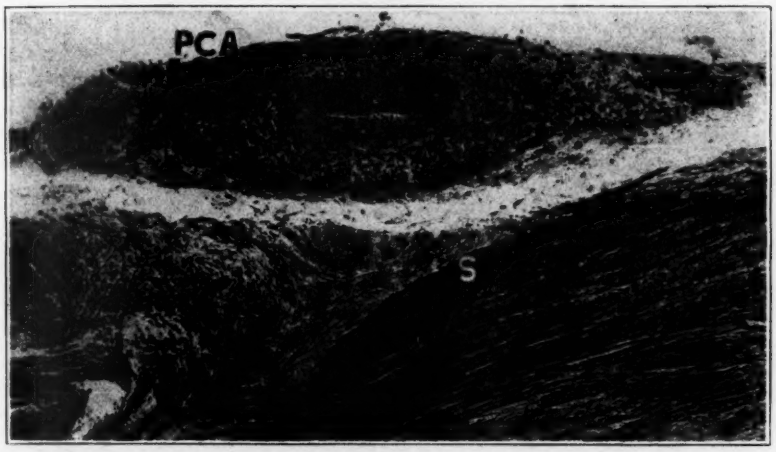

Fig. 2.—A short posterior ciliary artery, *PCA*, with highly infiltrated adventitia, media and intima; *S* indicates the sclera.

Fig. 3.—An infiltrated posterior ciliary artery, *PCA*, in longitudinal section, entering the layers of the dura, *D*, at the level of the lamina cribrosa.

Otherwise, there was moderate infiltration of the intervening structure with round cells, particularly about the blood vessels. There were a few evidences of hemorrhage in the tissue, due to rupture of the smaller vessels following fragmentation or occlusion by clots of fibrin. The capillary layer of the choroid and the elastic lamina were intact. The pigment epithelium showed little or no disturbance. The chromatophore system of the choroid was somewhat less dense than normal but showed no definite decrease in the number of cells or in their content of pigment.

The striking alterations of the retina were shrinking and fragmentation of the layer of rods and cones and its almost complete separation from the pigment

Fig. 4.—The lamina cribrosa, *L*, and the disk are richly infiltrated with round cells.

epithelium. In no section was there evidence of the normal continuity of these layers. Frequently there was accumulation of a granular exudate containing free pigment and pigment epithelium in the subretinal space (fig. 1 *C*).

In the episclera there was typical disease of the short posterior ciliary arteries, consisting of dense infiltration of all the layers, but without necrosis such as seen in the choroidal arteries. In serial section not a single vessel in this group was free from change. Similar infiltration was present in their course through the sclera and suprachoroidea. Of some interest was the disease of a vessel entering the dura and proceeding to the region of the lamina cribrosa. Such infiltration could actually be traced into the lamina and the nerve immediately in front. The optic disk contained many inflammatory round cells in addition to recently acquired glial tissue in the pit. The optic nerve was infiltrated behind the lamina and otherwise showed evidences of atrophy in its upper portion.

COMMENT

The lesions in this case correspond to those described in a great majority of the cases of this condition reported. Apparently the arteries of the ciliary system are most vulnerable. In almost every instance the choroidal vessels are affected and in some the short posterior ciliary arteries as well. In cases in which the retinal artery has been found to be diseased such change is limited in the portion behind the lamina cribrosa and not in the retinal arterioles. That the changes are restricted to these vessels conforms with the general rule that periarteritis nodosa attacks only vessels of medium size or those with well defined muscular coats. Von Herrenschwand expressed an interesting opinion regarding the site of predilection of the lesion. It appeared in his case that the periarterial nodes occurred particularly where the artery altered its direction, such as at its entrance into the sclera, its course through the lamina cribrosa and in its course from the sclera toward the ciliary body.

In the present case the posterior ciliary arteries were severely diseased, while those of the choroid were moderately so. It is fair to assume that the destruction of the layer of rods and cones and the extensive subretinal exudation are to be traced to disease in the choroid. As regards the appearance of the fundus, the widespread pigmentary disturbance may be traced to the disturbance in the neuro-epithelial layer and perhaps also to the subretinal exudate. In explanation of infiltration of the disk and the lamina cribrosa, there was sufficient indication that there had been neuritis which was dependent on disease of the ciliary arteries supplying this region (the circle of Zinn). Although the widespread disturbance of neuro-epithelium was undoubtedly a factor, it is extremely doubtful that even this could have produced such rapid loss of vision. There are, nevertheless, two distinct anatomic grounds for the loss of vision. It would appear, however, that the more rapid and greater loss is to be attributed to the local change in the nerve and that the retinal lesion was contributory. What would have ensued had the patient survived is problematic; perhaps detachment, as in Böck's last case, would have resulted, due to separation of the epithelial layer.

FITTING OF PROSTHESES FOR PATIENTS WITH CRYPTOPHTHALMOS AND EXTREME MICROPHTHALMOS

PAUL GOUGELMAN
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Since ophthalmologists are beginning to realize that it is possible to fit a prosthesis in cases of congenital blindness of one eye for cosmetic improvement, many patients, varying in age from 3 months to early adult life, have been referred for such fitting. Many patients handicapped by congenital blindness have gone through life unaware that it *is* possible to fit a prosthesis without the necessity of a surgical operation—the deformity may be overcome so that it is not evident. In fact, it has been my experience over a period of many years that in such cases the natural globe presents an ideal foundation for the introduction of a prosthesis.

Usually the globe is reduced in size, but it is far superior to any implant, so that the best cosmetic results are possible. In fact, the results are vastly superior to those in cases in which an excessively large implant is used, making the proper adaptation of a prosthesis impossible. The surgeon in his endeavor to produce a perfect foundation for the prosthesis often uses an implant that is too large and does not leave sufficient room for the artificial eye. There is no advantage in using a gold or any other implant 20 or 24 mm. in diameter that results in a stump as large as the natural eye. While the stump apparently is a fine one and almost as large as the good eye, it leaves no room for the prosthesis, and the result is sure to be a failure. Besides, a large implant invariably destroys the fold of the upper lid and produces apparent ptosis.

In cases of cryptophthalmos and extreme microphthalmos the stump is small, but as the muscles are intact it is possible to fit a proper sized prosthesis, which is replaced by progressively larger ones as conditions warrant. The fear of sympathetic ophthalmia has prevented many ophthalmologists from recommending the use of a prosthesis in such cases. In my experience, no ill effects have been seen in many hundred patients who were fitted. I do not know of any case in which it was necessary to remove the globe. Of course, such patients are fitted only on the recommendation of the ophthalmologist and when his examination demonstrates that the eye is quiescent and that no danger of sympathetic ophthalmia exists.

It has been the custom of many ophthalmologists in the past to discourage or at least defer the fitting of a prosthesis for an infant. Frequently the physician advises the parent to wait "until he is 7" or until some later period or advises against the procedure. In my opinion, this is a mistake, as lack of development takes place on the affected side, and frequently permanent malformation occurs. Instead of delay, immediate action is strongly advised, so as to prevent the deformation and shrinkage that is almost certain to result. A prosthesis should be inserted even in the case of very young infants.

In many cases if shrinkage is not too great the damage may be overcome if the person doing the fitting takes the time and understands

A, appearance of the patient before the prosthesis was fitted; *B*, appearance after the prosthesis was fitted.

the proper procedure in developing the lids and bony structures by fitting progressively larger eyes as the orbit enlarges commensurate with the general body growth. Care should be exercised, and progress should be made slowly. Sufficient time should be allowed between the various fittings so as to avoid the danger of irritation to the defective globe which might cause damage to it or retard the final result.

Two cases have recently come to my attention. One child has been fitted on the advice of the ophthalmologist since the age of 3 months, and each year a larger artificial eye has been inserted. At the present time no deformity is apparent. The mother of this child met a former schoolmate who had a child of the same age with the same abnormality, which had existed for about the same time, but she had been advised

to wait "until he was older." There was marked shrinkage on the side of the face with the shrunken globe, and the lids were tight and had lost their activity through disuse. It was difficult to handle this child, as he resisted any efforts to place an eye in the socket, and it took a great deal of time and perseverance to accomplish this. After the child's confidence was gained, by periodically changing the eyes a fairly good result was obtained, but it would have been much better had the first prosthesis been fitted in early infancy. Possibly as the child develops and a larger eye is fitted each year a normal-looking socket may be obtained.

A of the figure shows the appearance of a woman 26 years of age, before the fitting of a prosthesis. She had been advised early in life that it was not possible to fit an eye and went through life thinking this to be true. When she consulted an ophthalmologist as to the condition of the eyes of her child he inquired why she did not wear an artificial eye. She informed him that her parents had been advised that it was not possible. She was referred to me, and I found that she had a perfectly movable stump. After the orbit developed as a result of fitting with progressively larger eyes for six months, an excellent cosmetic effect was obtained. The motion of the eye was good, and no deformity was apparent.

In this case there was a marked psychologic effect on the patient due to the greatly improved cosmetic effect. The importance of advising a patient with anophthalmia to resort to a prosthesis is particularly evident in this case, as the future happiness and well-being of the patient was at stake. The change of expression seen in *B* of the figure is due not to the art of photography but to the prosthesis.

Another patient with apparent anophthalmia could not secure a position as stenographer or secretary at the age of 18 owing to her appearance and the psychologic effect produced by it. The ophthalmologist consulted early in life advised that she "wait until she is older." Fortunately, little shrinkage had taken place, and in a few months the socket was normal, permitting the use of a prosthesis that matched the good eye perfectly.

There was also a marked improvement in the patient's mental attitude and her outlook on life. She promptly went out to secure a position; she got it and all the other things in life that she had denied herself owing to self-consciousness induced by her deformity. She is thankful to the ophthalmologist who advised her that she could wear an artificial eye.

AN UNCLASSIFIED TYPE OF OPTIC NEURITIS

REPORT OF CASES

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The etiology of optic neuritis covers a wide range of known possible factors, but after these factors have been carefully eliminated there remains a group of cases in which no cause can be found. These cases we classify as instances of acute infectious optic neuritis.

In the last nine months we have observed seven such cases, in which the optic disks presented a swelling varying from 1 to 6 D. In all there was sudden loss of vision with central scotomas, and in two there was no perception of light. In one case, in which the patient was first examined in 1931, we made a diagnosis of optic neuritis of unknown cause. The patient returned in February 1937, showing advanced consecutive atrophy but vision of 20/30 in each eye. Another patient, who had sudden temporary loss of vision in 1932, came to us first in March 1937. She showed atrophy of the papulomacular bundle, with vision of 20/200 in each eye. The onset in the other five patients has occurred during the past nine months. Since October 1936, our Atlanta colleagues have observed five additional cases of optic neuritis of undetermined cause. The ages of the patients varied from 5 to 22 years. In no case during the course of the neuritis were there any general symptoms, but in most instances a prodrome of malaise, slight cold and sore throat antedated the onset from one to two months. Characteristic also were soreness and pain on movement of the eyeballs. Atrophy of the optic nerve occurred early and was consecutive in type (postneuritic); its severity varied with the duration of the swelling. If vision failed to show improvement early, i. e., in six weeks, the prognosis was grave. Lillie [1] has said that in general the prognosis of optic neuritis is poor, but in his group of miscellaneous unclassified cases it was particularly so. The pallor of the optic nerve was no indication of the visual acuity.

Read before the Section on Ophthalmology at the Eighty-Eighth Annual Session of the American Medical Association, Atlantic City, N. J., June 9, 1937.

1. Lillie, W. I.: The Clinical Significance of Retrobulbar and Optic Neuritis, Am. J. Ophth. **17**:110-119 (Feb.) 1934.

Review of the American literature for the past decade has failed to show any specific classification of these cases. In a study of forty-three cases of true optic neuritis seen in our private practice since 1932, none was found that we could place in this group.

In these seven cases there was no history of familial disease or of sinus infection, otitis media or trauma. The Wassermann reaction of the blood of all the patients and that of the spinal fluid of the five patients whose spinal fluid was examined was negative. Roentgenologic studies of the cranium and sinuses, hematologic studies and urinalyses gave negative results in all the cases. The results of general physical examination and special examinations of the ear, nose and throat were also negative. Each patient was essentially emmetropic

TABLE 1.—*Age and Sex of Patients*

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7
Age of patient, years	16	8	17	19	19	9	12
Sex	M	M	F	M	F	F	F

TABLE 2.—*Laboratory Findings of Consequence*

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7
Wassermann test of the blood	0	0	0	0	0	0	0
Wassermann test of the spinal fluid	0	0	0	0	—	0	—
Other tests of the spinal fluid	0	0	0	0	—	0	—
Roentgenogram of the skull	0	0	0	0	0	0	0
Roentgenogram of the sinuses	0	0	0	0	0	0	0
Red cells	0	0	0	0	0	0	0
White cells	0	0	0	0	0	0	0
Results of urinalysis	0	0	0	0	0	0	0

under the influence of a mydriatic. Finally each patient was examined by a competent neurologist (W. A. Smith, R. B. Wilson or E. F. Fincher), with, except for the optic neuritis, entirely negative findings.

REPORT OF CASES

CASE 1.—*History.*—W. W. B., a husky, well nourished boy, was referred to us on Sept. 16, 1936. The day after a week-end at an ocean resort about two months earlier he had complained that his eyes hurt and were watering. The next day, although a rather severe cold in the head developed, he returned to work. He was not particularly drowsy, and he had no fever, as far as he knows, no frank headache and no visual disturbance. After about two weeks the cold cleared up completely.

Five weeks before consulting us the patient had difficulty in reading because his eyes watered and the letters ran together. He continued to work for three days but felt that his vision was growing worse. He consulted an ophthalmologist, who told him that his vision was "about 10 per cent in the left eye and 20 per cent in the right." A general physical examination gave negative results. Then an otolaryngologist reported clinical evidence of sinus infection. Roentgen examina-

tion did not confirm this, and on draining the sinuses no pus was obtained. Enlarged tonsils were removed three weeks before we saw him. The boy thought he could see a little better after the operation and that he could see better with the right eye than with the left. He had had slight headaches at times since the onset of his illness.

At the age of 6 years, after a cut on his foot, he had been extremely ill for several weeks; his mother thinks that he was unconscious at times during this illness, but he had no convulsions.

Ophthalmologic Examination.—Vision of the right eye was 10/200, and that of the left was 5/200. The corneas were clear. The anterior chambers were normal. The pupils were half dilated (owing to the effect of a mydriatic). The media were clear, and the disks were oval, swollen and engorged and elevated about 2 D. There were very slight edema of the periphery and marked tortuosity of vessels. The maculae were normal, and no lesions were seen in the periphery of the fundus.

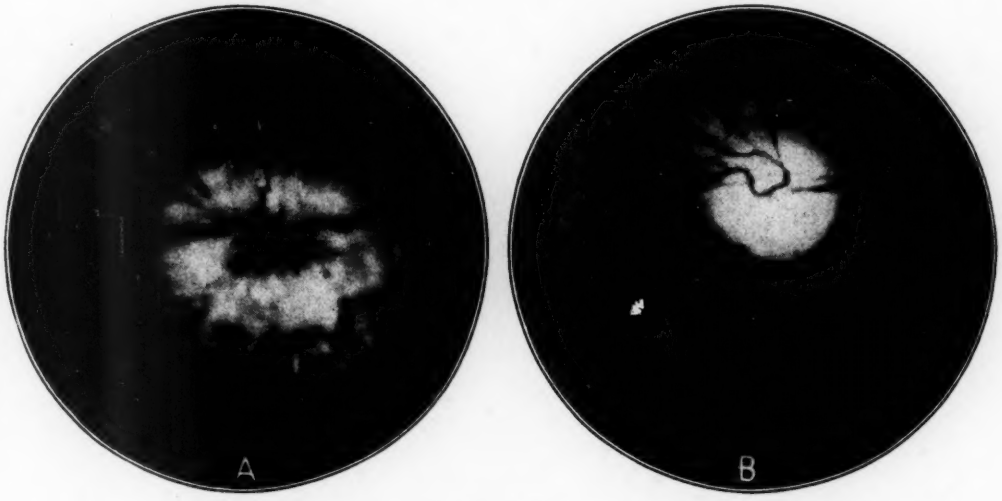

Fig. 1 (case 1).—*A*, photograph showing acute infectious optic neuritis with swelling of the disk of 2 D. *B*, photograph showing consecutive atrophy (six months after *A* was taken).

The visual fields were full peripherally but showed a central scotoma of 5 degrees in the right eye and of 10 degrees in the left.

Diagnosis.—The condition was diagnosed as acute infectious optic neuritis.

Treatment and Course.—The patient was put at absolute rest in bed. Typhoid vaccine (40,000,000 organisms) was given intravenously at intervals of five days. Fluids were limited to 1.2 liters a day, and 10 cc. of saturated solution of magnesium sulfate was given twice daily. One month later the swelling in the nerve heads had decreased to about 1 D., and the patient felt that he could see a little better. He was less nervous, and no new symptoms had appeared. Vaccine therapy was discontinued. In November the swelling in the nerve heads had practically subsided, but vision remained unchanged. He received four more injections of vaccine at five day intervals, with a considerable reaction, the temperature reaching 104 F. In January 1937 vision of the left eye had improved from 5/200 to 10/200. The right eye showed a reduction in the size of the

scotoma to 2 degrees. His general health remained good. In February there had been no great change, but the disks were fairly well outlined, and consecutive changes were beginning to appear. He was last seen in March. There had been no further improvement in vision. The atrophic change in the nerve heads was more advanced.

CASE 2.—*History.*—R. R. M., a fairly well developed, well nourished boy, was referred to us on Oct. 14, 1936. Ten days before he had complained of a headache; he had a slight cold, with disturbance in breathing at night. A few days later he experienced some pain over the left eye and on covering the right eye discovered that his left eye was totally blind. A physician was consulted immediately, but he found no abnormalities. The following day vision of the right eye began to fail, and when examined by us the patient was unable to distinguish light with either eye.

Tonsillectomy had been performed two years before, and he had had scarlet fever and pertussis in the past two years.

Ophthalmologic Examination.—The extra-ocular movements were normal in all the cardinal directions of gaze. The corneas were clear, and the pupils dilated evenly and were round. The lenses and media were clear. The nerve heads were swollen 4 D. The veins were enlarged; the arteries were somewhat tortuous, and there were a few hemorrhages over the nerve heads and some exudates adjacent to the nerve. The maculae were normal, and the remainder of the retinas showed no lesion. There was no perception of light.

Diagnosis.—The condition was diagnosed as acute infectious optic neuritis.

Treatment and Course.—The child was put to bed and given typhoid vaccine (20,000,000 organisms) intravenously; the temperature reached 103 F. This treatment was repeated every four days. Fluids were limited and saline cathartics given daily. After ten days there was some reduction in the swelling of the nerve heads. Vision had improved slightly, increasing to perception of light and color. Continuation of this regimen was urged, as the child was taken home. We have not been able to observe the boy since, but his father reports that his vision has not improved.

CASE 3.—*History.*—S. E. G., a well developed but slightly undernourished girl, was referred to us in February 1936. Four weeks previously she had begun to have burning and soreness in her lids. She had treated herself with boric acid solution but became no better. Two weeks later vision of the left eye began to blur, and the next day vision of the right eye became affected. She consulted her physician, who gave her a complete physical examination, with negative findings. Her tonsils were removed at this time "as a precautionary measure." Her vision remained blurred for the next week. Two days before we saw her she began to distinguish colors and large objects.

Her past history was essentially without significance except for frequent colds in the head during the past two years.

Ophthalmologic Examination.—Muscular movements were normal in the cardinal directions of gaze. Weakness of convergence was present. The pupils were round and dilated evenly. The lenses and media were clear. The nerve heads were swollen 1 D.; the veins were engorged, and the arteries were normal in caliber. The maculae were normal, and the retinas showed no pathologic lesion. A central scotoma of 20 degrees was present in each eye, and vision was 10/200.

Diagnosis.—The condition was diagnosed as acute infectious optic neuritis.

Treatment and Course.—The patient was put to bed and given three doses of typhoid vaccine (30,000,000 organisms) intravenously at four day intervals. She showed a marked reaction, the temperature reaching 104 F. after these injections. Fifteen days later the swelling of the pupilla had receded to a barely measurable amount. There were no signs of secondary changes. She was kept in bed, however, and given two more injections of vaccine. The reaction was not so severe. She was feeling much better, and thirty days after we first saw her the disks looked fairly good; rings were visible on the temporal sides. Vision was 20/200 in each eye. She was allowed to get up and resume moderate activity. Examination in April showed only slight residual changes in the nerve head; no new symptoms had developed. Vision was 20/70 in each eye.

Comment.—The tonsillectomy was performed at an opportune time, but the patient's physician reported that the tonsils showed no pus or inspissated material.

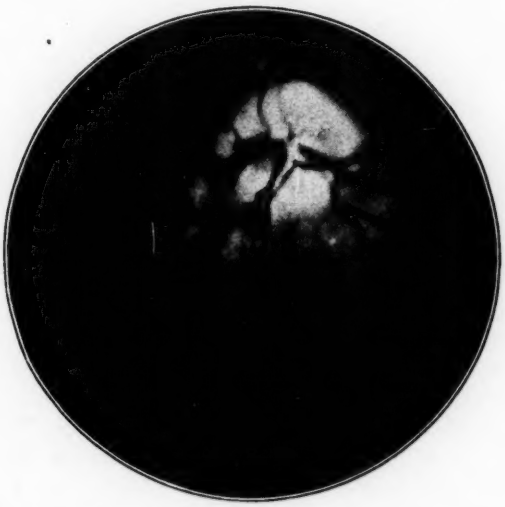

Fig. 2 (case 3).—Photograph showing acute infectious optic neuritis with swelling of the disk of 1 D.

Her sinuses were not operated on. There has been no change in her vision at this time, and the fundic picture remains the same.

CASE 4.—*History.*—R. H., a well developed, well nourished youth, was referred to us on Nov. 8, 1936. Ten days previously he had noticed dimness of vision in his left eye, which had progressed in three days to complete blindness. The next day he went blind in the right eye. He consulted his local physician, who sent him to us. The day following the onset of blindness he had pain in both eyes and rather severe headaches. Except for a mild cold in the head, he felt well.

Ophthalmologic Examination.—The ocular movements were well performed. The corneas were clear. The pupils were dilated (owing to the effect of a mydriatic). The lenses and media were clear. The disks were swollen 6 D. There were numerous flame-shaped hemorrhages over the disks, and the swelling extended somewhat into the adjacent retinas. The veins were engorged and slightly tortuous, but the arteries seemed normal. The macular areas were normal, and the peripheral part of the retinas showed no lesion.

Diagnosis.—The condition was diagnosed as acute infectious optic neuritis.

Treatment and Course.—The patient was hospitalized and given typhoid vaccine intravenously (20,000,000 organisms) every third day, the doses being increased to 170,000,000 organisms by the eighth injection. The general reaction was fairly severe, the temperature reaching 104 F. twice. After the third dose vision began to improve; objects could be distinguished, and the swelling began to subside. This improvement continued, and on December 1 vision was 20/40 in each eye. The nerve heads were swollen only 1 D.; the arteries and veins seemed normal, and the retinas and maculae were normal. The patient was dismissed from the hospital, still restricted in intake of fluids and told to resume partial activity. In December vision was 20/30 plus in each eye; the lamina cribrosa could be faintly seen, and the disks showed no beginning atrophic changes. No general symptoms have developed. In March 1937 his ophthalmologist reported that there had been no further change.

CASE 5.—*History.*—A. N. D., a well developed, well nourished young woman, consulted us in March 1937. She had applied for entrance in a nurses' training school, and routine examination showed deficient vision. There was no other complaint or symptom. In January 1932 she had been ill with influenza. A week after the onset and while she was still in bed she noticed decided blurring of vision in both eyes. This grew worse until she could barely recognize her family. This condition lasted only three days and then began to improve. She felt, however, that she had never been able to see so well after this; she always had trouble reading, although during the past two years her vision had improved.

Ophthalmologic Examination.—The ocular movements were well performed; the corneas were clear, and the pupils, which were 3 mm. in diameter, reacted to light and in accommodation. In each eye the lens and media were clear. The disk showed a decided temporal pallor, with blurred margins. The vessels were normal in size and relationship. The macula was normal, and the periphery showed no lesion. The visual fields showed central absolute scotoma of 5 degrees, with a relative scotoma of 10 degrees; examination was made with a test object of 5 degrees. Vision of each eye was 20/200.

Diagnosis.—The condition was diagnosed as consecutive optic atrophy with involvement of the papulomacular bundle. The primary cause, we feel sure, was acute infectious optic neuritis.

Comment.—This girl, of course, could not begin nurses' training. She has had no general symptoms since her illness in 1932; her health remains excellent. We believe that her condition is typical of acute infectious optic neuritis. The prognosis for any increase in her visual acuity is poor.

CASE 6.—*History.*—O. H., a well developed and well nourished girl, was seen first in April 1931. One week before, vision in her left eye had become blurred. There had been no previous serious illness. A slight cold had come on two weeks earlier. Two days before, her local ophthalmologist had found the left nerve head to be hazy and swollen and the right one normal. Vision of the right eye was 20/20, and that of the left 20/200.

Ophthalmologic Examination.—The right eye was normal. The pupil of the left eye dilated evenly and was round. The anterior chamber was normal, and the lens and media were clear. The nerve head was swollen 4 D. There was an apparent increase in the size of the capillaries over the disk. A small flame-shaped hemorrhage opposite 3 o'clock was present. The veins were tremendously engorged but showed slight tortuosity. The arteries were normal in caliber, and the macula was normal. The remainder of the retina showed no lesion.

Diagnosis.—A diagnosis of acute infectious optic neuritis was made.

Treatment and Course.—The patient was put to bed, limited in intake of fluids and given typhoid vaccine (20,000,000 organisms) intravenously at five day intervals for five doses and potassium iodide in doses of 5 grains (0.3 Gm.) daily. In May the swelling in the left nerve head had decreased to 2 D.; the hemorrhages had absorbed, and the venous enlargement was less. She was feeling physically well, but vision of the left eye remained the same. Three weeks later the swelling in the left eye was 1 D., and vision of this eye had improved to 20/70. Two days later she suddenly became blind in the right eye coincident with slight headache and malaise. There were accompanying visual hallucinations. Examination showed the right nerve head to be swollen 1 D. The swelling of the left nerve head continued to recede. She was put back to bed and given more typhoid vaccine in the vein at five day intervals. In July the swelling in the left eye had almost disappeared. There was definite consecutive atrophy present. The right eye showed a little swelling, with a return of visual acuity to 20/40. She was

Fig. 3 (case 7).—Photograph showing acute infectious optic neuritis with a stellate figure in the macula.

allowed to get up and resume activity, as no general symptoms developed during the course of the optic neuritis. She was not seen again until October 1934. She had had no illness in this interval; she had developed normally, and her eyes had been perfectly comfortable. She had had no trouble with her school work. Each disk was distinctly pale, especially the left one. Consecutive atrophy was present, but visual acuity of each eye was 20/30. In April 1936, the findings were unchanged. Visual acuity was 20/30 in each eye; the peripheral fields were full on examination with a 5 mm. test object, and no central scotoma was elicited. Her general health had been excellent.

CASE 7.—*History.*—F. F., a well developed, well nourished girl, was examined first in December 1932. One week earlier she had noticed failing vision in her left eye. There were no other symptoms. Her general health had always been good, there having been no serious illness in her lifetime. Her eyes had never been examined, but she read a great deal without difficulty.

Ophthalmologic Examination.—The right eye was normal. In the left eye the cornea was clear; the pupil dilated evenly and was round, and lens and media were clear. The nerve head was swollen some 5 D. There were numerous flame-shaped hemorrhages over the disk; the veins were greatly engorged, but the arteries were about normal in caliber. There were a few scattered exudates adjacent to the swollen nerve. The macular region showed a clearcut star. Vision of this eye was 20/200; that of the right eye was 20/20.

Diagnosis.—A diagnosis of optic neuritis and stellate retinitis was made.

Treatment and Course.—The patient was put to bed and given intramuscular injections of 8 cc. of boiled milk at four day intervals. There was a good reaction, the temperature reaching 102 F. Two weeks later the swelling of the nerve head had decreased to 2 D.; the macular star was still present, and the vision remained the same. The patient was feeling well, and no new symptoms had developed. She was allowed out of bed. Two months later she began to menstruate, and coincident with this she felt that her vision had improved. Vision at this time was 20/100 in the left eye and 20/20 in the right. The swelling in the nerve head had decreased to 1 D. The star figure in the macula was much less marked. She was allowed to resume full activity, but was instructed to limit her intake of fluid to 1.5 liter daily and to check carefully the daily elimination. She was seen again in 1933, at which time vision of the right eye was 20/20 and that of the left eye 20/70. The swelling had disappeared. Atrophic changes were present. The macular region was clear, and the star figure had entirely disappeared. She was examined again in 1934. Vision of the left eye was 20/50; that of the right eye was normal. Her general physical condition remained good; no new symptoms had developed, and the ocular findings had not changed.

Comment.—This condition was certainly acute infectious optic neuritis with the star figure in the macula as described by Fuchs.

GENERAL COMMENT

There is much confusion in the literature in distinguishing between optic neuritis and retrobulbar neuritis. Because of the proved central scotoma the condition in our seven cases would be classified by many as retrobulbar neuritis. In like manner, in most of the reported cases of optic neuromyelitis in which there was papillary swelling of 2 to 3 D. the condition has been diagnosed as retrobulbar neuritis. The same picture of a swollen nerve head and a central scotoma may be seen in acute syphilis of the central nervous system. We believe that retrobulbar neuritis should include only those conditions in which there is a normal fundus and that, regardless of the visual fields, every nerve head swollen because of inflammation represents optic neuritis.

It is possible that some cases of retrobulbar neuritis attributed to sinus disease in which recovery follows operation on clinically normal sinuses belong to this class. Benedict [2] expressed the opinion that foreign protein therapy would accomplish as much as surgical inter-

2. Benedict, W. L.: Retrobulbar Neuritis and Diseases of the Nasal Accessory Sinuses, Arch. Ophth. **9**:893-906 (Jan.) 1937.

vention on the sinuses unless they are clinically diseased. Perhaps a similar group of cases occurs with retrobulbar neuritis as the only finding.

The fundic picture in our seven cases resembles closely that found in the demyelinating diseases associated with optic neuritis, especially neuromyelitis optica. The acute onset, the reaction and swelling of the nerve head, the central scotoma and the inevitable consecutive atrophy give a picture parallel to that of neuromyelitis optica save that the cord is not involved. It should be repeated that in the course of time the disk may take on the clearcut marginal aspect of primary atrophy: in our case the picture could easily have been mistaken for primary atrophy had the neuritic history not been known.

The sudden onset and rapid loss of vision lend weight to the theory that in these cases the condition was of infectious origin. The number of cases in this group occurring so closely together makes us feel too that the disease has epidemic characteristics. Wilson [3] stated that he has seen more cases of acute myelitis with recovery in the last nine months than in the previous seven years. Is it not possible that the same causative agent is present in these cases but with a different specificity?

Axial neuritis extending into the lamina cribrosa certainly must occur, the involvement being confined to the optic nerve, as there is no clinical evidence of involvement of contiguous tissue. The virus (if it be one) that is responsible could easily and most likely does belong in the class of large viruses or micromicrobes.[4] There is a growing opinion that multiple sclerosis is not caused by a neurotrophic virus and that the so-called cases of acute multiple sclerosis are really instances of acute encephalomyelitis or encephalitis. We realize that the condition in our cases could be mistaken for a first episode of multiple sclerosis. In multiple sclerosis, however, the early visual disturbances are usually slight and transitory, and the illness does not seem serious enough for the patient to consult a physician until general symptoms appear. The first evidence of visual disturbance in a proved case of multiple sclerosis is usually elicited in the history, and the eye is objectively normal at the time of examination. In the experience of Rivers and his collaborators,[5] there has been no proof of demyelinization being produced by any known neurotrophic viruses. Demyelinization prob-

3. Wilson, R. B.: Personal communication to the authors.

4. Rivers, T. M.: Recent Advances in the Study of Virus and Viral Diseases, J. A. M. A. **107**:206-210 (July 18) 1936.

5. Rivers, T. M.; Sprunt, D. H., and Berry, G. P.: Observation on Attempts to Produce Acute Disseminated Encephalomyelitis in Monkeys, J. Exper. Med. **58**:39-53 (July) 1933.

ably did not occur in our cases, but, unfortunately, we have no proof of the true pathologic change.

The treatment of the patients of our series has been similar; it was, in general, empirical, as we were dealing with an unknown cause. Foreign protein in varying amounts was used, care being taken to produce a moderate systemic reaction with elevation of temperature to from 102 to 104 F. The response seemed somewhat in proportion to the severity of the reaction produced. We advise complete rest and quiet for at least two weeks in all cases of this condition and longer, if necessary, depending on the response to treatment.

In one patient (case 7) who showed a stellate figure in the macula along with the neuritis the condition possibly offers a clue to the etiology of Fuchs' idiopathic retinitis.

CONCLUSIONS

We feel that the condition in these seven cases warrants a new classification, namely, acute infectious optic neuritis, the cause being an unknown virus with predilection for the optic nerve.

ABSTRACT OF DISCUSSION

DR. E. L. GOAR, Houston, Texas: The knowledge of the subject of optic neuritis is in a rather unsatisfactory state. The nomenclature, the pathogenesis, the etiology—none of these is thoroughly defined or well understood. In Duane's last translation of "Fuchs's Text-Book of Ophthalmology" (1923) papilledema is still discussed under the heading of optic neuritis. The terms retrobulbar neuritis, axial neuritis and toxic amblyopia are all used more or less synonymously. To add further to the confusion, the term interstitial neuritis has been injected, designating in this connection inflammation of the fibrous septums of the nerve, which are probably the only parts of the nerve ever primarily affected in true inflammatory lesions.

I propose that the situation be clarified by having a common understanding of the terms used, somewhat as follows: Retrobulbar neuritis should be used for a condition in which there is sudden loss of vision, with a central scotoma, but no visible pathologic changes when the eye is viewed with the ophthalmoscope. Toxic amblyopia should be used for a condition in which there is gradual loss of vision, with a central or a paracentral scotoma, which may be relative or absolute, and with no ophthalmoscopic evidence of inflammation, and which is due to some known toxin. In optic neuritis there may be any or all of the signs mentioned for the other conditions, but there must be inflammation of the disk visible with the ophthalmoscope. Papilledema, or choked disk, is a noninflammatory swelling of the disk due to increased intracranial pressure.

When one attempts to trace the etiology of the condition, the case is far more complicated. Almost every illness the human body is heir to at one time or another has had to bear the onus of optic neuritis. Syphilis, meningitis, meningo-encephalitis, sinus disease and multiple

sclerosis are all known and frequent offenders. Infections of the teeth or tonsils, diabetes, pellagra, and poisoning, such as that with lead, alcohol or nicotine, are less prominent, but they have all been indicted, along with other factors too numerous to mention. And now a filtrable virus is offered as the offending agent. I am not sure on what grounds the authors arrived at this conclusion, but certainly if all unclassified cases fall in this group, it will be fairly large. A filtrable virus has been proved to produce herpetic keratitis and has been suspected of causing sympathetic ophthalmia, and it is not beyond the bounds of reason to believe that it may attack the optic nerve.

I must say one word about sinus disease and optic neuritis. Lillie, in writing on the subject of optic neuritis a few years ago, stated that among five hundred cases of optic neuritis observed at the Mayo Clinic, in only one was the condition attributed to sinus disease. No doubt some years ago ophthalmologists were entirely too enthusiastic about sinus disease as a cause of optic neuritis, but I am convinced that the pendulum has swung too far the other way. When one sees well defined optic neuritis in a patient with purulent infection of the sphenoid sinuses or the posterior ethmoid sinuses and when the sinuses are drained and the neuritis begins to improve forthwith, that is about as good evidence of cause and effect as one is able to establish by clinical methods, even if it does savor slightly of reasoning on the principle of post hoc, ergo propter hoc. I have seen this happen in far more than 0.2 per cent of cases.

Neuromyelitis optica is a rare disease. According to Perrit's statement in 1934, only a little over fifty cases had been reported, five of them in the English literature. The disease is characterized by bilateral optic neuritis, either acute or subacute, following, as a rule, but occasionally preceded by, or simultaneous with, myelitis. As one reads the description of the disease in the reports of the cases, it seems doubtful whether this should be classed as a clinical entity or as an episode in one of the inflammatory or degenerative diseases of the central nervous system.

The cases reported by the authors resemble those classed as instances of the ocular phase of neuromyelitis optica, but the essential of the cord involvement is lacking. The solution of the problem of the etiology of what the authors have well named this condition, acute infectious optic neuritis, probably awaits the discovery of the causation of other puzzling diseases of the central nervous system, such as poliomyelitis, Parkinson's disease, multiple sclerosis, encephalitis and others, consideration of which has occupied the best minds among the neurologists for years, but which is still undetermined. Meanwhile, the virus theory offers an attractive field for speculation and experimentation.

Dr. Walter I. Lillie, Philadelphia: I had the privilege of going over Dr. Clay and Dr. Baird's paper in detail. The report of the seven cases shows that the visual loss was precipitous in onset and remained stationary for a long period. After therapy was instituted, gradual improvement was noticeable. To me, this verifies the opinion that the prognosis for optic neuritis is much more grave than that for retrobulbar neuritis. Usually the patients with retrobulbar neuritis will get well as one is sending them around to the various physicians to obtain an opinion as to what the cause might be. Optic neuritis

persists, owing, I think, to the marked swelling of the fibers of the optic nerve in the lamina cribrosa impinging against the more or less inelastic sclera, so that even a small amount of inflammation may produce tremendous damage.

The etiology in these seven cases is a moot question, because the patients fall in the age group of those with multiple sclerosis.

My associates and I had the same experience with cases that later proved to be instances of multiple sclerosis, although the first examination and subsequent examinations gave negative results after the original episode of optic neuritis.

It was also true in my experience that cases of this type after a number of years did not turn out to be instances of multiple sclerosis; so there is a small group in which the etiology cannot be definitely established.

This type of optic neuritis may occur at any age. It occurs most frequently in the age group given, but at the present time I have a patient, a man aged 74, who is in the hospital with bilateral acute optic neuritis. The nerve heads are elevated 2 D., and the patient is totally blind. He had no neurologic changes whatever, but for the past three days bulbar palsy has been present. Whether this patient belongs to the group with acute optic myelitis I do not know, but the condition might readily come under this classification.

The condition in the seventh case of Dr. Clay and Dr. Baird is very interesting because it has been given a separate name by Fuchs, stellate retinitis associated with optic neuritis. I saw a boy aged 14 who had sudden loss of central vision in each eye. This had been present about four weeks before I saw him. The slide shows swelling of the nerve heads with an incomplete macular star in each. To me, this means that this patient has had previous edema in the macular region, which is now undergoing absorption. In other words, it is a good criterion, I think, from a prognostic standpoint that the condition is getting better, and it needs no separate classification as stellate retinitis.

Dr. J. M. Baird, Atlanta, Ga.: I should like to thank Dr. Goar and Dr. Lillie for their kind remarks in discussing this paper. We realize that this subject is very controversial, but we felt, after making an exhaustive study of each of the patients and obtaining no positive findings, that this was probably the best classification we could think of for a diagnostic or disease entity.

HUMAN FACTOR IN AIRPLANE CRASHES

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AND
G. RAND, PH.D.
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There seems to be a growing conviction that the pilot is an important factor in the increasing number of airplane crashes.[1] There is perhaps a good reason for this conviction. It may be that with the rapid improvement in the facilities for aviation the attitude of the pilot has changed with respect to the importance of his own qualifications, fitness and training and to the highly specialized nature of the services he was formerly called on to contribute and still has to contribute in emergencies. It may be also that not enough attention is paid to fitness in the selection of pilots and to making sure that they are in fit condition for service at all times when they are called on to render service. It seems strange that the plane should be carefully tested on every point of its construction and operation before each flight and little or no attention given to the pilot at that important time other than to see that he is on hand to fly. With the advance in scientific and mechanized control the importance of the human factor has without question been thrust into the background.

During the World War we devised an instrument for testing the fitness of aviators. This instrument was used at Mineola, N. Y., for testing and studying the fitness of candidates for entrance into the air service and later taken to France by Dr. William Holland Wilmer, surgeon in charge of the Medical Research Laboratories, Air Service, American Expeditionary Force, for the purpose of studying the fitness of aviators already in the service for the various tasks required of them, particularly in combat flying. Since the war the instrument has been

From the Research Laboratory of Physiological Optics.

1. Major-General James E. Fechet (Ret.), former chief of the United States Army Air Corps, for example, who has devoted a great deal of time to the study of airplane crashes, stated (Flight Surgeon Topics, School of Aviation Medicine, Randolph Field, Texas, April 1937, vol. 1, p. 44) that in more than half the number of cases these crashes are due to an error on the part of the personnel or to undetermined causes. In the personnel group he includes the following: the pilot, the weather man, the manager of the airline operations and the mechanic. Of these, the pilot is of course an important factor. A small percentage of these crashes, less than five, he said, are due to mechanical failures—malfunction of an engine or breakage of some part of the plane or of its essential accessories. A considerably higher percentage are due to bad weather—ice, fog or storm.

greatly improved and is now being manufactured by the Gaertner Scientific Corporation, of Chicago. As manufactured by the company the instrument has been still further improved. One of the earlier improved models was described in a previous number of the ARCHIVES.[2] In 1933-1936 this model of the instrument was used by Lieutenant-Commander C. J. Robertson, of the United States Naval Service, for the study of fitness for aviation on such points as entrance requirements, disqualification for the service on account of age, fatigue in relation to the number of hours in the air, individual susceptibility to fatigue and other points. Dr. Robertson has published his results in a series of articles in the *United States Naval Medical Bulletin* and the ARCHIVES.[3]

With this instrument can be measured, among other things, the speed of change of adjustment of the eyes for clear seeing at near to clear seeing at far and back again to near. This involves a measurement of the speed of vision, the speed of use of the muscles of the eyes in the perfect coordination needed for the clear seeing of a small detail and the speed of accommodation. It constitutes an extremely sensitive test of the ocular and oculomotor fitness of the aviator and of small disturbances in this fitness and also a delicate and effective test for bodily and mental fatigue and other disturbances in physical and mental facility and proficiency. Fatigue, for example, has to be tested through its effect on some function. Perhaps no more delicate means can be found for detecting fatigue than observation of its effect on speed in those uses of the eyes which require highly coordinated changes in muscular adjustment. The delicacy and accuracy of coordination that are required in these adjustments will be realized when one remembers that changes in the convergence of the eyes are made by six pairs of muscles which serve to support as well as to move the eyes, and that the breadth of the images on the two retinas which must be combined into one in seeing is for the standard test object of the order of thousandths of a millimeter. Also, in changing the vision from near to far and back again to near, the muscles of accommodation must act in perfect coordination with the muscles that move the eyes. Still further, the sensorium must function at a high level of efficiency.

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2. Ferree, C. E., and Rand, G.: An Instrument for Measuring Dynamic Speed of Vision, Speed of Accommodation and Ocular Fatigue, Arch. Ophth. **15**:1072 (June) 1936.

3. Robertson, C. J.: Measurement of the Speed of Adjustment of the Eye to Near and Far Vision, U. S. Nav. M. Bull. **32**:275 (July) 1934; A Comparative Study of the Measurement of the Speed of Adjustment of the Eye for Near and Far Vision, ibid. **33**:187 (April) 1935; Measurement of Speed of Adjustment of Eye to Near and Far Vision, Arch. Ophth. **14**:82 (July) 1935; Measurement of Speed of Adjustment of Eye to Near and Far Vision: A Further Study, ibid. **15**:423 (March) 1936; Effect of Fatigue on the Adjustment of the Eye to Near and Far Vision, ibid. **17**:859 (May) 1937.

The test is without doubt one of the most sensitive that has ever been devised for the detection of any imperfection in the oculomotor, accommodative or sensory functions or any temporal disturbance in these functions. It is with these temporal disturbances that we are particularly concerned in this paper. Common causes of these disturbances are fatigue, loss of sleep, worry or any mental state which distracts attention, the variations in physical and mental efficiency and alertness common to every one in the course of time and illness. Any of these may be sufficient to cause the aviator to fail or falter at a critical time in the high degree of service that is required of him. The profound effect of fatigue and other disturbances of bodily and mental efficiency on such highly organized and delicate muscular coordinations as are required in the speedy use of the eyes is too well recognized to need further mention here.

It seems strange that so much care is taken to see that the plane is in perfect condition before a flight is undertaken and so little attention given to the condition of the aviator. While it is true that a human being cannot be treated as a machine, it is known, as has already been noted, that he is subject to many disturbances from day to day that render him unfit for services which require supernormal fitness and proficiency and involve responsibility for human life and safety. It seems only reasonable, therefore, that the fitness of the aviator, as well as the fitness of the plane which he operates, should be tested before each flight is undertaken. It is surely not enough to require only an entrance test of fitness and then allow him to go on without further check, even without regulation of his conditions of living, until age or some mishap retires him from service.[4]

Our personal feeling is that a test of each aviator should be made immediately before and after each flight. We offer the following reasons for this:

1. The test before the flight can be used to prevent the aviator from going into the air when he is clearly and dangerously unfit for service. It is neither fair nor good public policy that knowledge of

4. We understand that some improvement in this respect has already been made or is in contemplation. The following statements, for example, are quoted from the article by Major-General Fechet referred to earlier in the paper: "Striving to promote continued pilot fitness, we developed a new profession in the Army—that of Flight Surgeon. He has paid us handsome dividends. We found that annual or semi-annual examinations were not enough. We needed a smart medico to keep the pilots under daily observation. . . . Flight Surgeons paid off in reducing airplane crashes. I commend that thought to commercial airline operators." He said further: "Health is mental as well as physical. I think the mental side plays a bigger rôle in air pilot health than the physical. A man who is worried and preoccupied about domestic discord or financial extremities may be more unsafe than one subject to fits or fainting spells."

his fitness should depend on his own report. In combat flying, in particular, he might well be prevented from making such a report through fear of being called a slacker or because of patriotism or personal pride. In commercial service, too, many reasons might operate to deter him from making a report of unfitness. The responsibility for making such report should be taken out of his hands and consigned to a competent examiner. A surgeon, however long his experience and however well demonstrated his ability, volutarily subjects himself to a test of steadiness of hand and keenness of eye before undertaking a critical operation. Surely in these offices requiring services equally responsible for life and safety there should be some test of fitness immediately before the service is undertaken. Such precautions might be considered extreme had it not been so clearly demonstrated that something is radically wrong in modern aviation.

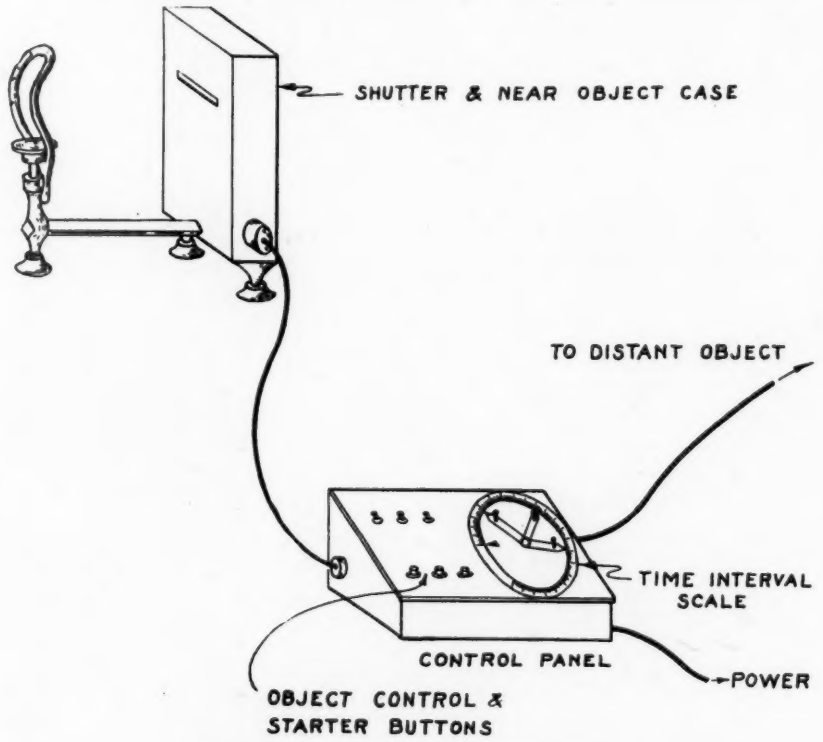
2. The test at the end of the flight would indicate how well the aviator has stood the strain of his service. It would give valuable information as to his susceptibility to fatigue and make it possible to assign him to the length and kind of service he is capable of performing. It would also give a great deal of valuable general information as to the number of hours in the air and the amount of strain which aviators, taken collectively, can reasonably be expected to stand.

3. From the results of the tests, graphs or curves can be plotted which will give a splendid picture of the aviator's fitness, his endurance, his susceptibility to fatigue, the consistency of his service and other points. In short, these records would serve as the basis for a high type of personnel service in aviation. From these graphs it can also be readily seen when the aviator is becoming incapacitated for service through age or some other cause. This alone should be a sufficient reason for adopting some such program.

4. A feasible test with a suitable instrument is available. The test does not require more than ten minutes to perform, and the result can readily be given a numerical rating. The instrument is easy and convenient to operate, and the entire program is well within the technical capabilities of the average flight surgeon.

Two forms of the test may be suggested: (1) The time required for the discrimination of the object at near and for the change to far and back again to near may be measured in each test, or (2) in a series of preliminary tests the median or average time required for these performances may be determined for each aviator and taken as his standard of performance. In the routine procedure of testing, the instrument should be set to give these times of exposure. The test may consist of ten or some suitable number of trials to ascertain in what percentage of cases the aviator can attain his standard of performance. This percentage may be accepted as the index of his fitness at that time.

The instrument recommended may be called a multiple exposure tachistoscope. As described in the previous issue of the ARCHIVES,[2] it comprises, it will be remembered, a driving mechanism, four sectored disks so arranged and of such size as to expose in immediate succession, in turning, a near test object on the left, a far test object in the median plane and a near test object on the right. The test objects are the letter E, the openings of which can be turned in eight different directions to give an objective check on the judgment. The far test is provided



An electrical multiple exposure tachistoscope.

with a remote control such that these adjustments can be made by pressing an electric key at the position of the examiner. The distance of the far test surface and the lateral separation of the two near test surfaces can be varied at will. The sectored disks are turned by means of a constant speed motor provided with gears to give suitable reductions in speed and an intermittent gear which causes the disk covering the test objects prior to the beginning of the exposure to stop in exactly the same position at the end of a single rotation. In the preferred form the values of open sector are adjusted by small worm gears which serve both to change the position of the movable disks and to hold them firmly in position for any given setting. Exposure may be made in

continuous series up to four seconds. Both the angular values of open sector and the times of exposure can be read from suitably positioned, graduated scales.

A later form of the instrument is an electrical tachistoscope. This is the form now being manufactured by the Gaertner Scientific Corporation. In the figure are shown the control panel and the case containing the near test objects, the means for illuminating these test objects and the shutters for exposing both the near and the far test objects. The shutters are operated electromagnetically and are so arranged as to expose in sequence the test objects in the order already noted, namely, the near test object on the left, the far test object in the median plane and the near test object on the right. In the control panel are housed relay devices for operating the shutters and adjusting the position of the far test object. On the outside of the panel are provided suitable buttons and switches for these controls and a dial or time interval scale with hands that can be set to give the values of exposure desired. This form of the instrument has been designed especially to give compactness of construction and the maximum convenience of operation.

Either of the aforementioned forms of the instrument makes possible the following determinations: (*a*) the use of a set of very sensitive tests, which take into account as no other tests do both the motor and the sensory functions of the eyes in just the proportion that they occur in the act of seeing objects in different directions and at different distances; (*b*) the testing of the dynamic speed of vision with either the oculomotor or the accommodative feature emphasized (in the dynamic test for speed of vision the eyes are required to shift their regard from one object to another or to a series of objects and to discriminate them in turn during the time or times of exposure; these conditions test not only the speed of reaction of the sensorium but also the oculomotor facility and proficiency), and (*c*) the measurement of the time required to change from near to far and from far to near in combination or separately.

The test with this instrument has the following practical uses: (*a*) It is a means of detecting abnormalities and depressions in the oculomotor functions in the work of the clinic. (*b*) It constitutes a test for vocational fitness in all cases in which dynamic speed of vision is an important requirement. (*c*) It may be used as a limiting test for age. (*d*) It is a means of measuring ocular and oculomotor fatigue and recovery and of testing individual susceptibility to ocular fatigue and the capacity to recover. Because of the profound effect of fatigue and other disturbances of bodily and mental efficiency on such highly organized and delicate muscular coordinations as are required in the speedy use of the eyes, the test may be used also as a very sensitive means of detecting bodily and mental fatigue and other disturbances

in physical and mental proficiency which occur frequently in the normal course of living. It may be used also for detecting and studying the effect of change of altitude, temperature and other variations in the physical conditions to which the aviator is subjected. (*e*) It may serve as a means of training eyes to greater oculomotor and accommodative facility.

SUMMARY

With the growing conviction that the pilot is an important factor in the increasing number of airplane crashes, it seems that more attention should be paid to fitness in the selection of pilots and to making sure that they are fit for service at all times when they are called on to render service. It is strange that so much care is taken to see that the plane is in perfect condition before a flight is undertaken and so little attention is given to the condition of the pilot. While it is true that a human being cannot be treated as a machine, it is known that he is subject to many disturbances from day to day that render him unfit for services which require supernormal fitness and proficiency and involve responsibility for human life and safety. These disturbances can be shown by test.

In the paper a very sensitive test for these disturbances in fitness is proposed, and a convenient instrument for giving the test is briefly described. The test falls well within the technical capabilities of the average flight surgeon and should not require more than ten minutes to perform. It is recommended that the test be given both immediately before and immediately after each flight. The reasons for this are as follows: 1. The test before the flight can be used to prevent the aviator from going into the air when he is clearly and dangerously unfit for service. It is neither fair nor good public policy that a knowledge of his fitness should depend on his own report. The responsibility for making such a report should be taken out of his hands and consigned to a competent examiner. 2. The test at the end of the flight would indicate how well the aviator has stood the strain of his service. It would give valuable information as to his susceptibility to fatigue and make it possible to assign him to the length and kind of service he is capable of performing. It would also give a great deal of valuable general information as to the number of hours in the air and the amount of strain which aviators, taken collectively, can reasonably be expected to stand. 3. From the results of the tests, graphs or curves can be plotted which will give a splendid picture of the aviator's fitness, his endurance, his susceptibility to fatigue, the consistency of his service and other points. From these graphs it can also be readily seen when the aviator is becoming incapacitated for service through age or some other cause.

NEAR REACTION OF THE PUPIL IN THE DARK

A QUANTITATIVE STUDY

F. HERBERT HAESSLER, M.D.
MILWAUKEE

Although pupillary reactions have been thoroughly (one would be tempted to say exhaustively) studied, I have found no reference to a publication of data on the effect of adjustment for near vision acting alone as a stimulus to pupillary contraction. As is well known, the pupil contracts when the illumination of the eye is increased and also when the pair of eyes is adjusted for near vision. To study the character of the latter reaction alone it is necessary to observe the effect on the size of the pupil of near vision in darkness. This was accomplished as follows: In a totally dark room the subject of the experiment fixed his gaze on one of the luminous characters on the dial of a radiolite watch which was held at a measured distance from the eye and in such a position that the angle between the axis of the eye and the camera remained constant throughout. The shutter of a previously adjusted camera was then opened, and a flash bulb the illumination time of which was less than one-tenth second was ignited. It is known that there is a latent period of one-fifth second in the pupil's reaction to light; hence the picture records the size of the pupil which results from the near adjustment only. That the light of the radiolite watch is below the threshold of the pupil for light can be demonstrated by the entoptic pupillary phenomenon. Figure 1 makes clear the practical arrangement of this simple apparatus, and figure 2 shows how a constant angle can be maintained between the axis of the eye and that of the camera.

The apparatus consists of a head and chin rest (*a*), a Leica camera (*b*), a socket for the flash bulb (*c*), and a card with the fixation object *e*, movable on the bar *d*. The bar *d* has notches in it, which can be felt in the dark, so placed that the angle between the visual axes when the eye fixes an object at these points will be 6, 12, 18, 24 and 30 degrees, respectively. It was assumed that the center of rotation of the eyeball is 12 mm. behind the corneal apex, and a separate stick was prepared for each subject, the measurements being based on the interpupillary distance.

In each experiment the subject placed his head firmly in the head and chin rest. The camera and the rod carrying the fixation object were

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adjusted satisfactorily. The room was darkened, and five minutes was allowed for the eyes to become dark adapted. A picture was then taken with the eyes focused on the object located at a distance such that there was an angle of 6 degrees between the visual axes. All further manipulations were easily performed in the dark. After two or three minutes, when the after-image of the flash-light had paled, the radiolite object was moved to the next notch, and a flash-light photograph of the same eye was taken again. This process was repeated until photographs were obtained of the pupil in the state which it assumed when the angle between the visual axes was 6, 12, 18 and 24 degrees, respectively. These distances were chosen in the belief that the near reaction of the

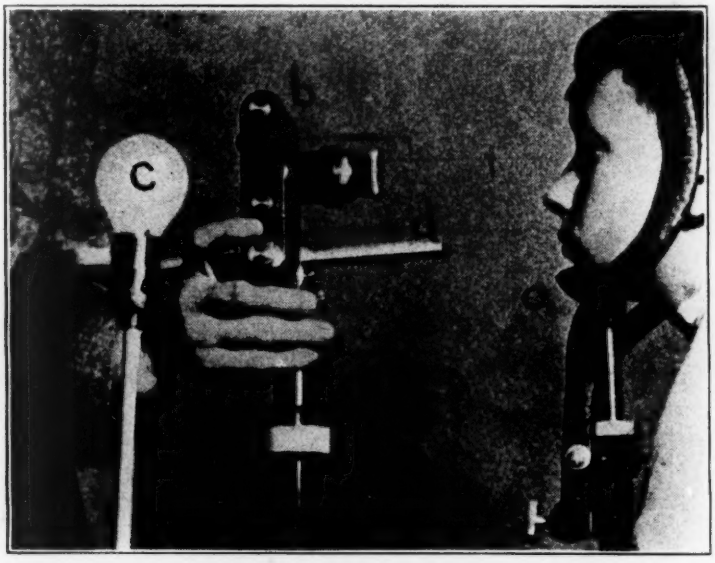

Fig. 1.—Apparatus for study of the near reaction of the pupil in the dark. *a* is the head and chin rest; *b*, the camera; *c*, the flash bulb, and *e*, the fixation object, movable on the bar *d*.

pupil is probably associated with convergence rather than with accommodation. If this is true the graphs for the two processes could be represented by straight lines, which, with the introduction of a suitable constant, would be parallel. The graph showing the accommodation for these distances expressed in diopters is a curved line, but it differs so little from the graph for convergence that it is not possible to correlate the curves for the pupillary changes with one or the other of them. The pupillary and corneal diameters were measured directly on a projection from the film enlarged so that the image of the cornea was from 8 to 10 mm. in diameter.

All the data gathered are presented. No photographs were ignored except the first few taken in experiments to try out the technic. Once the series was started, all the data were considered. The subjects were a boy of 11 years, two girls aged 12 and 13, respectively, and a woman aged 36. In refraction carried out with the pupils under the influence of homatropine the refractive error was found to be less than 0.5 D. The subjects had no hyperphoria, and their eyes could converge beyond the point necessary to make the angle of 24 degrees. Inspection of the data makes it clear that the decrease of the pupillary diameter is proportional to the increase in the angle of convergence in twenty-seven

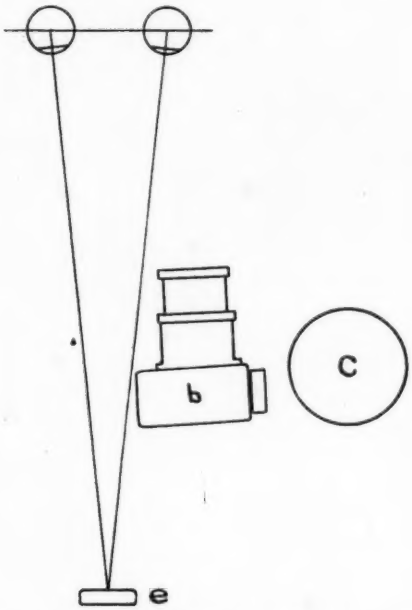

Fig. 2.—Diagram illustrating the principle of the apparatus. *b* is the camera; *c*, the flash bulb, and *e*, the fixation object.

of the forty experiments. The figures in the third, fourth, fifth, sixth and tenth columns state the measurement in millimeters of the pupillary and corneal diameters on the projected image. In the seventh, eighth and ninth columns the decrease in the pupillary diameter from step to step is recorded in millimeters as measured on the projection. In the eleventh and twelfth columns are stated the maximum and the minimum pupillary diameter in millimeters and tenths of a millimeter, as calculated from the assumption that the actual corneal diameter of all the eyes was 11 mm.

In the twenty-seven series of photographs of the pupil which I have considered successful, no one increment of shortening differs from the

average of the series by more than 20 per cent. As the pupillary circumference is proportionate to the pupillary diameter, the decrease in the pupillary diameter represents shortening of the fibers of the sphincter pupillae. Inspection of the table reveals no uniform tendency among the thirteen series of photographs that differ from the majority; there-

Data for Experiments

Experiment	Subject	Difference Between Relative Pupillary and Corneal Diameters, Mm.				Increment of Change, Mm.			Relative Corneal Diameter, Mm.	Pupillary Diameter, Mm.		Normal Near Reaction
		6°	12°	18°	24°					Maximum	Minimum	
1	H	49.0	45.5	42.0	38.0	3.5	3.5	4.0	80	6.7	5.2	+
2	H	47.5	44.0	40.0	36.0	3.5	4.0	4.0	78	6.7	5.1	+
3	L	43.0	38.5	35.5	35.5	4.5	3.0	0.0	78	6.1	5.0	..
4	L	39.0	36.5	31.0	28.0	2.5	5.5	3.0	79	5.4	4.9	..
5	L	42.0	40.0	34.0	31.0	2.0	6.0	3.0	79	5.8	4.3	..
6	H	47.5	43.5	39.5	35.5	4.0	4.0	4.0	80	6.5	4.9	+
7	H	48.5	45.0	41.0	37.5	3.5	4.0	3.5	76	7.0	5.4	+
8	H	48.0	44.5	41.5	38.0	3.5	3.0	3.5	76	6.9	5.5	+
9	H	51.0	47.0	42.0	32.0	4.0	5.0	10.0	79	7.1	4.5	..
10	L	44.0	35.0	31.0	29.0	9.0	4.0	2.0	80	6.0	4.0	..
11	L	40.0	36.0	32.0	28.0	4.0	4.0	4.0	78	5.6	3.9	+
12	A	50.0	46.0	42.0	38.0	4.0	4.0	4.0	80	6.9	5.2	+
13	A	46.0	43.0	41.0	38.0	3.0	2.0	3.0	79	6.4	5.3	..
14	H	46.0	43.0	40.0	36.5	3.0	3.0	3.5	80	6.3	5.0	+
15	D	38.0	35.0	32.0	29.5	2.5	3.0	2.5	81	5.2	4.0	+
16	D	38.0	33.5	29.0	25.0	4.5	4.5	4.0	80	5.2	3.4	+
17	D	36.0	32.0	28.0	24.0	4.0	4.0	4.0	80	4.9	3.3	+
18	H	55.0	50.0	45.0	40.0	5.0	5.0	5.0	92	6.6	4.8	+
19	H	51.0	46.0	41.0	36.0	5.0	5.0	5.0	78	7.2	5.1	+
20	H	51.0	47.5	43.5	40.0	3.5	4.0	3.5	78	7.2	5.6	+
21	A	50.0	47.0	44.0	40.5	3.0	3.0	3.5	78	7.0	5.7	+
22	A	49.0	47.0	45.0	36.5	2.0	2.0	8.5	78	6.9	5.1	..
23	A	49.0	46.0	43.0	39.5	3.0	3.0	3.5	78	6.9	5.5	+
24	D	35.0	30.5	27.0	24.0	4.5	3.5	3.0	74	5.2	3.6	..
25	D	36.0	36.0	32.0	26.0	0.0	4.0	8.0	74	5.3	3.9	..
26	L	44.0	41.0	38.0	35.5	3.0	3.0	2.5	78	6.2	5.0	+
27	L	42.0	38.5	35.0	32.0	3.5	3.5	3.0	78	5.9	4.5	+
28	L	42.5	39.5	36.0	32.0	3.0	3.5	4.0	78	6.0	4.5	+
29	D	37.0	31.5	26.0	24.0	5.5	5.5	2.0	74	5.5	3.6	..
30	L	41.0	38.5	36.0	29.0	2.5	2.5	7.0	78	5.8	4.1	..
31	D	38.0	34.5	31.0	27.5	3.5	3.5	3.5	74	5.6	4.1	+
32	D	36.0	33.0	30.0	27.0	3.0	3.0	3.0	74	5.3	4.0	+
33	D	38.0	35.0	32.0	28.5	3.0	3.0	3.5	74	5.6	4.2	+
34	D	39.5	36.0	33.0	29.5	3.5	3.0	3.5	75	5.8	4.3	+
35	D	41.0	37.5	34.0	30.5	3.5	3.5	3.5	77	5.8	4.3	+
36	L	40.5	36.0	33.0	28.0	3.5	3.0	5.0	78	5.7	3.9	..
37	L	39.0	36.5	34.5	30.0	2.5	2.0	4.5	78	5.5	4.2	..
38	A	50.0	45.5	41.5	37.5	4.5	4.0	4.0	80	6.9	5.1	+
39	A	48.0	43.5	39.0	35.0	4.5	4.5	4.0	80	6.7	4.8	+
40	A	49.0	45.0	40.5	36.0	4.0	4.5	4.5	80	6.6	4.9	+

fore one can conclude, I think, that the twenty-seven series which are similar within the limits stated represent a description of the normal near reaction of the pupil in the absence of light.

SUMMARY

By means of instantaneous photographs of the pupils of the eyes that are in a known state of convergence in the dark, measurements were obtained which describe the pupillary reaction that is associated with near vision in the absence of other stimuli.

ABSTRACT OF DISCUSSION

DR. FRANCIS H. ADLER, Philadelphia: Dr. Haessler has studied the effect on the pupil of the near point reaction, that is, the combined action of accommodation and convergence for objects placed at known distances in front of the eye. He has undertaken these measurements with the retina partially dark adapted. In this respect these measurements are new, and the data constitute interesting information.

A number of authors have previously studied the effect of accommodation acting alone on the contraction of the pupil under conditions of illumination varying from 200 to 0.5 lux. One author studied the contraction of the pupil at different distances when the light was kept constant at high intensities of illumination and then subsequently studied the same reaction when the illumination was stepped down.

Dr. Haessler has carried this experiment one step further by reducing the illumination to practically zero and studied the near point reaction in a dark adapted retina. The results give information as to the effectiveness of accommodation and convergence on the tone of the pupil when all light is excluded from the eye, and, as I read his figures, they appear to prove that even when light is excluded from the eye the effect of accommodating and converging for a known distance, such that the angle between the visual axis is 6 degrees, is sufficient to exert a tone on the pupil to contract it somewhat, because his figures at the start of the test show a pupil of approximately 6 mm. in diameter, whereas the diameter of the pupil of a nonaccommodating and nonconvergent eye under similar conditions of dark adaptation should be higher than this.

One can be certain that convergence was carefully controlled in his measurements, because he gives the actual angle between the visual axes. One cannot be so certain that accommodation was equally carefully controlled.

I could not predict how much accommodation the subject was actually making when fixing on the radiolite dial of the watch at known distances. I wish to know whether the subject was required at each test to tell the figures on the watch and if the figures on the watch were sufficiently small to require an accommodative effort.

I would not agree, therefore, that what was found is due to convergence alone, but perhaps that is a minor and more academic question, since, as Hess and others have shown long ago, this reaction of the pupil is an associated reaction and not a reflex, and under experimental conditions both convergence and accommodation can be isolated, and each is successful in causing contraction of the pupil.

DR. FREDERICK H. VERHOFF, Boston: I should like to ask Dr. Haessler if he made any attempt to see whether adduction alone has an effect on the pupillary reaction. I do not see how any one could determine whether accommodation alone or convergence alone has an effect on the pupil, because I know of no experiment by which one can separate these two functions. Even if one covers one eye and looks at a near object, convergence occurs. If Dr. Haessler or any one else has a method of separating the effects of these two functions I should like to know what it is. In other words, it seems to me impossible to determine whether the pupillary reaction comes primarily from convergence or from accommodation.

In regard to adduction, it would be interesting to know whether or not the mechanism for this is entirely distinct from that of the mechanism for voluntary convergence, and it seems to me that Dr. Haessler might have been able to obtain data on this question by producing convergence by prisms and ascertaining whether or not the pupillary reaction differed under these conditions from that resulting from normal convergence.

Dr. F. Herbert Haessler, Milwaukee: Dr. Adler mentioned accommodation. An effort was made to be sure that the subject did accommodate clearly.

The figures on the watch are very small. The subject's attention was constantly called to a definite number, the implication being "You are sure you see the number?" The reply was: "Yes, I see the number." Also, in the experiment with the greatest angle of convergence, namely, that around 24 degrees, if one tries to increase that angle of convergence, in other words, bring the watch nearer to the eye, the subject can no longer accommodate. Under such conditions the subject complained of blurring of a very small image.

Dr. Verhoff's suggestion about separating accommodation and convergence within the limits of these experiments has not been followed. At the distances which give the angles of convergence chosen, the graph for accommodation is pretty close to that for convergence. It is not precisely a straight line, but the curve is so slight that the experimental data are too gross to enable one to decide this point. However, experiments have been reported in which the pupillary reaction has been shown to be definitely associated with the convergence reflex.

EQUAL ADVANCEMENT AND RECESSION OPERATION FOR HORIZONTAL STRABISMUS

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Based on the principle of equal advancement and recession of the opposing muscles for horizontal strabismus, the method of operation here described has been found dependable in giving accurately the planned alteration of position of the eyeball which preliminary examination and testing have shown to be desired. One muscle is to be receded the same number of millimeters that the antagonist is advanced. Because of the equal advancement and recession there is little or no tension on the fixing sutures during the operation or during the healing process; adhesion of the displaced tendons takes place at the site of the sutures. Following the described sequence of the steps of the operation contributes to securing this lack of tension of the sutures and consequently to the exactness of the result.

Measurement of the displacement of the muscles is made with calipers set for the desired distance in millimeters and used to measure both advancement and recession (fig. 1). Seven millimeters is the maximum distance practicable, and this is used only in cases of extreme deviation; if greater effect is needed it should be obtained by operation on the other eye. I have used only ordinary calipers with fine but blunt points, but those described by Dr. John Green, having one point sharp to anchor at the insertion while the other marks the sclera, would give additional accuracy.

The muscle to be advanced is exposed by a horizontal incision of the conjunctiva and Tenon's capsule, and its tendinous portion is freed from side attachments. A double-armed suture is placed exactly at the insertion of the tendon, its loop embracing the middle third of the width of the tendon together with a little of the superficial portion of the sclera (fig. 2 *A*). The two suture needles are passed under the upper and the lower border, respectively, of the tendon and through the tendon at the borders of its middle third at exactly the distance from the loop in the insertion measured by the calipers (fig. 2 *B*). The two sutures are then passed through the upper and the lower lip, respectively, of the incision in the capsule and conjunctiva at the level of the insertion and in line with the loop of the suture (fig. 2 *C*). The main advancement suture is in place before the muscle itself is disturbed, and its position can be precise (fig. 2 *D*). The advancement is now

interrupted for the purpose of doing the recession. This much of the advancement is done first because the deviated position of the eye gives the best possible exposure of the field of operation.

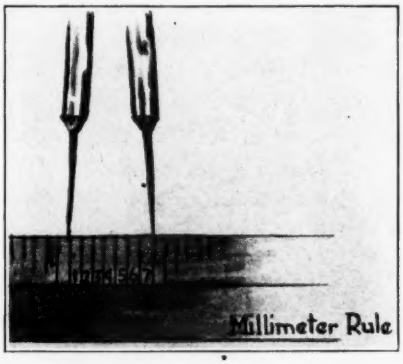

Fig. 1.—Calipers set for the desired change of position of the eye, to measure both advancement and recession; 7 mm. is the maximum distance.

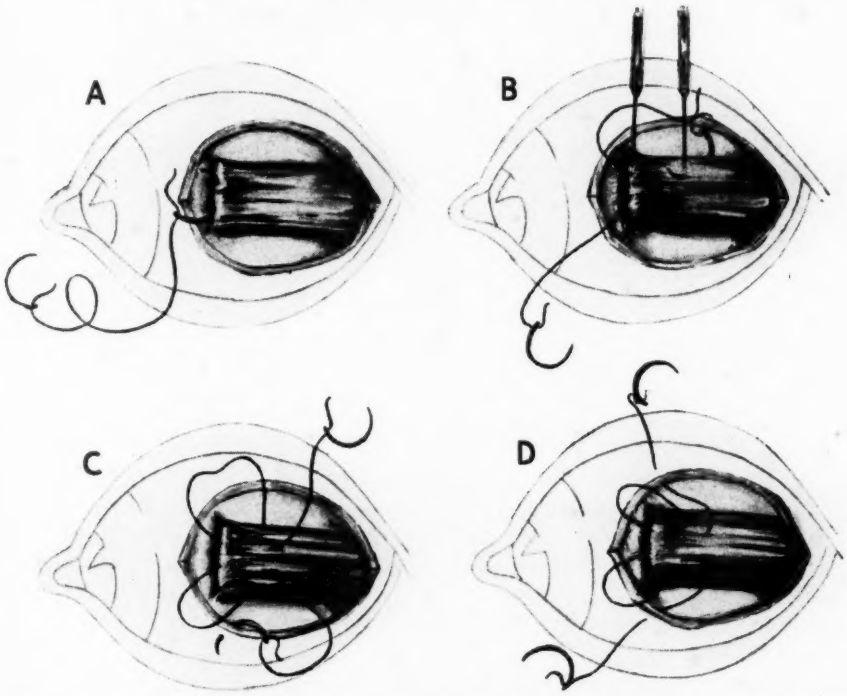

Fig. 2.—The muscle to be advanced is exposed by a horizontal incision of the conjunctiva and Tenon's capsule; a suture is placed at the insertion of the tendon, embracing the middle third of the tendon (*A*). The two suture needles are passed through the tendon at the borders of its middle third at the position measured by the set calipers (*B*). The two sutures are passed through the lips of the incision of the capsule and conjunctiva in line with the loop of the suture in the insertion (*C*). The main advancement suture is entirely in place before any alteration of position of the muscle (*D*).

The muscle to be receded is exposed by a horizontal incision of the conjunctiva and Tenon's capsule, freed from lateral attachments and raised on a thin and straight squint hook placed close to the insertion (fig. 3 *A*). The usual squint hook is too thick and too curved for this purpose. A double-armed suture is placed immediately behind the thin hook, its loop on the under-surface embracing the middle third of the width of the tendon, a little less than 2 mm. from the insertion. The two suture needles then are passed through the upper and the lower lip, respectively, of the incision in the capsule and conjunctiva, posterior to the suture loop, the distance being measured by the calipers (fig. 3 *B*).

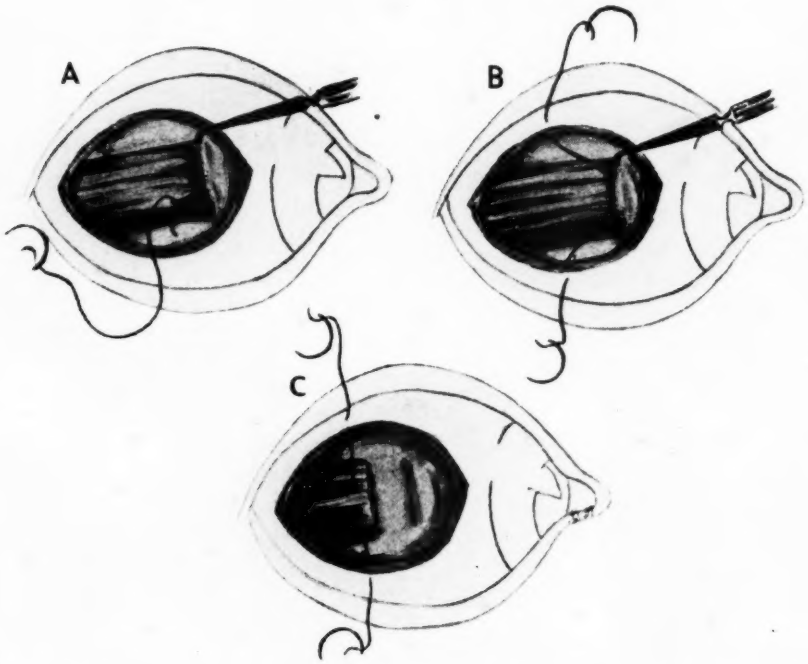

Fig. 3.—The muscle to be receded is exposed by a horizontal incision of the conjunctiva and Tenon's capsule; a suture is placed close to a thin hook, embracing the middle third of the tendon (*A*). The two suture needles are passed through the lips of the incision of the capsule and conjunctiva, the distance from the suture loop being measured by the calipers (*B*). The muscle, cut at its insertion in the sclera, retracts; the suture then is tied and secures the tendon to Tenon's capsule and closes the incision of the capsule and conjunctiva (*C*).

This measurement is made from the suture loop, not from the insertion. The tendon is cut from the sclera at the insertion, the hook remaining in place to protect the suture; this is done best with the Weber probe-pointed canaliculus knife. The severed muscle retracts, and the suture when tied unites the tendon with the capsule and conjunctiva (fig. 3 *C*). An additional suture may be needed to close the anterior

half of the conjunctival incision. It is essential that there be no vertical cuts or tears in the lips of the incision of Tenon's capsule, since the position of the retracted muscle during the process of healing depends on the integrity of the capsule between the suture and the capsule's anterior attachments. It is probable that adhesion of the tendon to the sclera almost always occurs finally; if not, the muscle is all the better for being lengthened rather than set back on the sclera.

The recession being completed, the advancement is resumed (fig. 4*A*). A second advancement suture is placed in the superficial portion of the sclera, with a vertical loop, 2 or 3 mm. anterior to the insertion of the main suture (about half the measured advancement). The

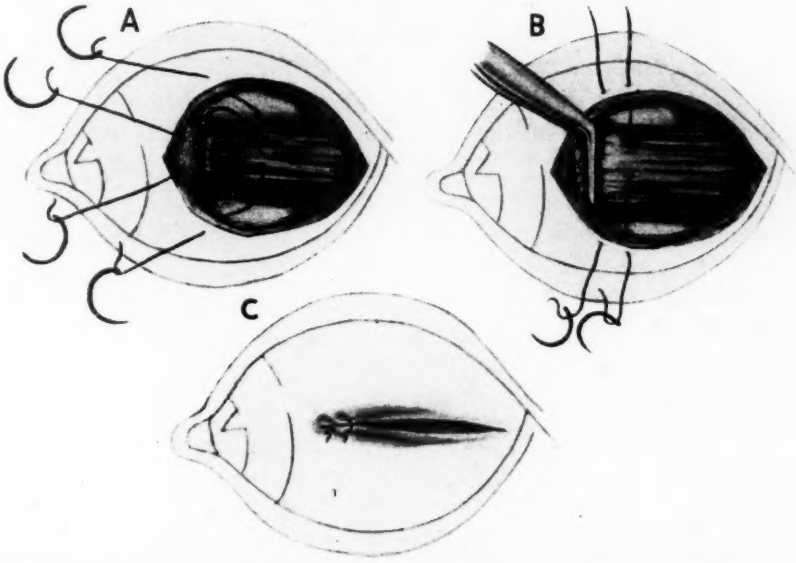

Fig. 4.—A second advancement suture is placed in the superficial portion of the sclera anterior to the main suture loop; the tendon, in the grasp of a muscle forceps, is severed between the forceps and the insertion (*A*). The tendon, in the grasp of the forceps, is elevated and drawn forward; the two needles of the anterior suture pass through the tendon at the borders of its middle third posterior to the forceps and then through the lips of the incision of the capsule and conjunctiva, being thus parallel to the main suture (*B*). The two advancement sutures, placed tandem and parallel, unite the sclera, tendon, capsule and conjunctiva and close the external incision (*C*).

tendon is grasped with a muscle forceps close to the insertion but with room left to cut the tendon between the forceps and the suture loop, care being taken to avoid cutting the suture; this too is done best with the canaliculus knife. The muscle in the forceps is elevated, and the two needles of the anterior suture are passed at the borders of its middle third, behind the forceps and 2 or 3 mm. anterior to the corre-

sponding part of the posterior suture, then through the upper and the lower lip, respectively, of the incision of the capsule and conjunctiva and again anterior to the corresponding part of the anterior suture (fig. 4 *B*). If the advancement is not more than 4 mm. a temporary suture loop can be used in place of the muscle forceps. The muscle is drawn forward to the advanced position; the sutures are tied, the posterior one being tied first, and the tendon end is tucked inside the incision after being trimmed with scissors if redundant. The advancement sutures, placed tandem and parallel, embrace in their loops the superficial portion of the sclera (the stump of the tendon is held in the posterior suture), the middle third of the tendon and both lips of the incision of the capsule and conjunctiva (fig. 4 *C*). The recession having been done, little traction is needed to draw the muscle into the position of advancement, and there is little tension on the sutures during the tying or during the healing. An additional suture may be needed to close the posterior half of the conjunctival incision. A binocular dressing is applied and maintained four or five days, with daily change for cleansing and use of any antiseptic which seems needed. Catgut sutures are left to absorb; silk sutures are allowed to remain undisturbed as long as eight days. General anesthesia is necessary for young children; for older children and for adults a 2 or 4 per cent solution of procaine hydrochloride injected along the two muscles is adequate.

CHEMISTRY OF THE RETINA

IV. THE BACILLARY LAYER

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Little is known about the chemical nature of the visual purple, the chief light-sensitive substance in the retina. In the past the retina with its ten or more layers was generally extracted with a solution of digitonin or of sodium cholate to give a colloidal solution of visual purple which contained many other chemical substances naturally occurring in the tissue. The crude extract was then examined by optical methods to determine its photochemical properties. Recently the whole retina was extracted with solvents of lipids, and carotenoids were found. The controversial reports on the carotenoids gave only slight evidence of their chemical nature. It is questionable whether or not the light-sensitive substance and carotenoids come from the bacillary layer or the rest of the retina or from both.

This paper is a report on the attempt to isolate and to determine some of the chemical properties of purified visual purple. The experience which was gained in the investigation of the constitution [1] and of the lipids [2] of the retina was a great aid in the treatment of this light-sensitive substance.

EXPERIMENTAL PROCEDURE

Extraction of Visual Purple.—Fresh eyes from cattle from 1 to 2 years old were kept cold and were not exposed to light. The retinas without the retinal pigment epithelium were immediately removed under a 10 watt red photographic light and were then ground in a mortar and suspended in a cold 1 per cent solution of sodium chloride, 1 cc. of solution being used for each retina. After centrifugation at 2,100 revolutions a minute for fifteen minutes a layer of dark red visual purple formed a ring separating the water-soluble material from the insoluble residue. The red substance was removed by decantation, and the residue was again extracted. The slimy red substance was freed of water-soluble material by washing with a 2 per cent solution of sodium chloride at p_H 7.8. The yield of the red substance, now called crude visual purple, from moist bovine retinas was repeatedly found to be about 2.7 per cent, calculated on the basis of dry weight.

Serial extraction of the layer of visual purple obtained from 200 bovine retinas was made (475 mg. calculated as dry weight). With a 2 per cent solution of digitonin about 400 mg. of crude visual purple was extracted, which contained 300

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1. Krause, A. C.: The Chemistry of the Retina: II. Chemical Constitution, Am. J. Ophth. **7**:555, 1936.

2. Krause, A. C.: The Lipids of the Retina, Acta ophth. **12**:372, 1935.

mg. of protein and 100 mg. of lipid. The material that was found to be insoluble after repeated extractions with solution of digitonin was extracted with a solution of sodium glycocholate until no soluble substance was obtained. The solutions of glycocholate contained 10 mg. of protein and 5 mg. of lipid. The residue was repeatedly extracted with a solution of sodium desoxycholate. The combined extracts contained 4 mg. of protein and 1.5 mg. of lipid. The insoluble residue consisted of a black pigment and a light gray gel. It contained 30 mg. of protein and 13 mg. of lipid. About 0.24 mg. of phosphorus was found in this lipid.

Visual purple dissolved in a 2 per cent solution of digitonin. About 1 mg. of sodium glycocholate and about 0.5 mg. of sodium desoxycholate, respectively, in aqueous solution dissolved 1 mg. of visual purple. Digitonin gave a pinkish red solution, sodium glycocholate a rose red solution and sodium desoxycholate a deep red solution, with the same concentration of visual purple. If the solutions are saturated with sodium or magnesium sulfate the visual purple is precipitated. A solution of sodium cholate dissolved little or no visual purple.

A series of samples each containing 1 cc. of a suspension of fresh visual purple layer which was equivalent to 19 mg. of the dried material were mixed with 1 cc. of a 2 per cent solution of digitonin, 1 cc. of a 2 per cent solution of sodium glycocholate and 1 cc. of a 2 per cent solution of sodium desoxycholate, respectively. After stirring for thirty-minutes, a red residue was found in the first two solutions. If the amount of red color of the solution of sodium desoxycholate was taken as 100, that of the solution of sodium glycocholate was 77 and that of the solution of digitonin was 43. However, repeated extraction removed the red color of the residues.

Analysis of Visual Purple.—A typical analysis of the crude visual purple, which was performed repeatedly, is described as follows:

The crude visual purple was separated from 123 Gm. of moist retina. It was repeatedly extracted with a 2 per cent solution of sodium glycocholate until the total volume of red extract was 50 cc. The gray gelatinous residue weighed 378 mg. and consisted of 308 mg. of protein and 70 mg. of lipid. The extract was half saturated with sodium sulfate at 25 C. A small white residue of lipoprotein, containing 158 mg. of protein and 24 mg. of lipid, was removed by filtration. The solution was extracted with small portions of purified petroleum benzine (petroleum ether) until nothing further was removed. The extract contained 10.4 mg. of lipid, of which 4.7 mg. was cholesterol.[3] It did not react positively to antimony chloride or for the presence of glycocholate. The solution was saturated with sodium sulfate, and the visual purple was precipitated. The visual purple was exhaustively extracted with alcohol and then with ether. The white residue consisted of 294 mg. of protein. The orange-colored extract contained 142 mg. of lipid, 273 mg. of glycocholate and 2.2 mg. of phosphorus. The extract was evaporated to dryness and dissolved in 50 cc. of chloroform. The chloroform solution was evaporated almost to dryness, and then 100 cc. of acetone was added. The mixture was stirred with a stream of nitrogen. The precipitate from the acetone solution was washed with small portions of cold acetone. It weighed 150 mg. and contained 2.14 mg. of phosphorus, 60 mg. of glycocholate and, qualitatively, choline.[4] The phosphorus

3. Man, E. B., and Peters, J. P.: Gravimetric Determination of Serum Cholesterol Adapted to the Man and Gildea Fatty Acid Method, with a Note on the Estimation of Lipoid Phosphorus, J. Biol. Chem. **101**:685, 1933.

4. Lintzel, W., and Monasterio, G.: Eine Methode zur Mikrobestimmung des Lecithins in Blute und Plasma, Biochem. Ztschr. **241**:273, 1931.

and choline were derived from the lecithin. After saponification with a 0.4 per cent solution of sodium hydroxide at 25 C. for thirty minutes, 30 mg. of colorless fatty acids, 0.2 mg. of cholesterol and less than 1 mg. of unsaponifiable lipid were found. The saponifiable and unsaponifiable fraction of the residue gave no reaction to antimony trichloride.

The soluble acetone fraction was evaporated to dryness. It weighed 53.5 mg. After saponification with 0.4 per cent solution of sodium hydroxide at 25 C. for thirty-five minutes, the nonsaponifiable part weighed 31 mg. and contained 8.0 mg. of cholesterol as determined by the colorimetric method and 7.6 mg. as determined by the digitonin method. After the removal of the cholesterol by digitonin and then the removal of the digitonin, a biphasic test was made on the residue, of which about 8 mg. dissolved in the 92 per cent methyl alcohol and 5.5 mg. dissolved in purified petroleum benzine. The golden yellow epiphasic residue gave a positive reaction to antimony trichloride. The colorless hypophasic residue showed no color with antimony trichloride.

The epiphasic golden yellow pigment of the crude visual purple gave a lemon-colored solution with purified petroleum benzine and a light red color with chloroform. In other solvents of lipids the color deepened according to the refractive index of the solvent. The pigment behaved like a carotenoid hydrocarbon in that it absorbed iodine, gave a positive (blue) reaction to antimony trichloride, passed to the epiphase, contained no nitrogen and formed a layer on calcium hydroxide in the chromatographic tube.

From the spectroscopic measurements it was found that about 65 per cent of the golden pigment is in the layer of visual purple and about 35 per cent in the rest of the retina. Since the amount of the retina without the visual purple is relatively large and the crude method of separation of the visual purple removes only the densest red layer, some of the remaining retina is slightly pink. If the golden yellow pigment is considered to be in a concentration of 100 per cent in the layer of visual purple as it is removed, the relative concentration of the golden pigment in the remainder of the retina is 5 per cent. It is probable, then, that the golden yellow pigment comes wholly from the bacillary layer.

The saponifiable fraction contained a yellow lipid which was insoluble in water but at p_H 7.5 or above became soluble and colorless in an alkaline solution. Acidification of the alkaline solution liberated the yellow lipid again. It gave a positive reaction to antimony trichloride and had an odor more like that of clupanadonic acid than like that of geronic acid or its derivatives. This yellow lipid pigment behaved like the golden yellow pigment except that it was preferentially absorbed by calcium carbonate and was precipitated by barium and silver salts and decolorized by alkalis. No other lipid pigments or lipids giving a blue reaction to antimony trichloride were found with the exception of very small quantities of vitamin A.

Action of Light on Visual Purple.—The following analyses were made to determine the action of light on the visual purple:

A 50 per cent saturated solution of sodium sulfate containing visual purple previously extracted with purified petroleum benzine was filtered and exposed to sun-

light for thirty seconds until the red solution turned clear golden yellow and was then extracted with purified petroleum benzine; the extract gave a positive reaction to antimony trichloride. A solution of visual purple was extracted consecutively three times with purified petroleum benzine, giving 4.5, 1.6 and 1.2 mg. of lipid, which contained 1.93, 1.03 and 0.93 mg. of cholesterol, respectively. After extraction this solution on exposure to light and subsequent bleaching of the visual purple gave up to purified petroleum benzine 10.5 mg. of lipid containing 2.5 mg. of cholesterol. This lipid gave a positive reaction to antimony trichloride, in contrast to the negative reaction of lipid obtained from the petroleum benzine extract from visual purple kept in the dark. In other words, light liberated a lipid containing free cholesterol precipitable by digitonin and another lipid giving a positive reaction to antimony trichloride. The nonsaponifiable fraction of the soluble acetone extract of the visual purple, which was exposed to light, gave a slight reaction to antimony trichloride, and the quantity of nonsaponifiable lipid was equivalent to 26 mg. The lipid contained 7.4 mg. of cholesterol. The crude yellow acid lipid of the saponifiable acetone extract weighed 13 mg.

If the golden yellow solution which was first exposed to light thirty seconds and which was then extracted with purified petroleum benzine was illuminated two minutes more, it lost 70 per cent of its yellow color as measured colorimetrically, and a light yellow precipitate formed. The precipitate consisted of 22 per cent lipid and 78 per cent protein. After a short time the solution became colorless and clear. If the golden yellow solution was made acid with acetic acid, a yellow precipitate and a clear solution resulted. After the precipitate was washed with slightly acidulated water, the solid was extracted with cold alcohol, a white residue of protein and a yellow solution of lipid being left. The yellow precipitate obtained by precipitation in acid was found to be 27 per cent lipid and 73 per cent protein. Alkalis changed the golden yellow alcoholic solution of lipids to a pale yellow solution from which 20.4 per cent of the lipids were extractable with purified petroleum benzine. This nonsaponifiable fraction contained 50.5 per cent cholesterol as determined colorimetrically and gravimetrically, but no nitrogen or phosphorus. The reaction to antimony trichloride was negative.

The nonilluminated retina, from which the layer of visual purple was removed, was treated in the same manner as the layer of visual purple. It was first extracted with purified petroleum benzine. The lipid residue weighed 1.3 mg. It gave no reaction to antimony trichloride and contained 0.8 mg. of cholesterol. The acetone insoluble fraction was found by analysis to be composed of large amounts of nucleic acid, lecithin, other complex lipids and glycocholate. Of 2.69 Gm. of total solids from the retina, 56 mg. of lipid soluble in acetone was found. After the saponification of the acetone soluble fraction no yellow carotenoid behaving like an acid was found in the 30 mg. The reaction to antimony chloride of the saponifiable fraction was slightly positive. In the nonsaponifiable fraction of 26 mg., 19 mg. of cholesterol and 4 mg. of a lipid which gave a slightly positive reaction to antimony chloride were found.

These analyses have been repeated many times on bovine visual purple during the past two years, and no essential differences were found in the results or in the color of the products.

The manipulation of the retina and extraction were done under red light and in the cold. The solvents and other chemical compounds

which were used were purified by standard methods and kept in the dark. The alcohol was free from aldehydes and acids. The purified petroleum benzine, with a boiling point of from 30 to 60 C., was free from residue and unsaturated hydrocarbons. The salts of the cholic acids were recrystallized and the composition checked by analysis.

COMMENT

The visual purple seems to be a complex lipoprotein consisting of protein and a lipid behaving like a carotenoid. A second lipid, with the properties of a highly unsaturated acid, which may be a product of decomposition, was found in the crude visual purple. On exposure to light a golden yellow lipid with the properties of a hydrocarbon carotenoid is liberated from the lipoprotein. Further exposure to light changes the golden lipid to a colorless lipid which has no carotenoid properties. This lipoprotein, or the visual purple, seems to be found only in the bacillary layer of the retina. The liberation of free cholesterol by light may occur from the breakdown of other retinal proteins of the bacillary layer.

Although no beta carotene or other carotenoid was found in the retina, there is no reason why it should not occur here, particularly in the retinas of the Jersey or Guernsey breed of cattle after grazing in the pasture. The report of Brunner, Baroni and Kleinau,[5] and that of Euler and Adler[6] that beta carotene is the chief carotenoid forming a part of the visual purple was not confirmed. Wald[7] was also unable to find carotene or xanthophyll in the bovine retina. There is a good possibility that either of the yellow lipids may act like vitamin A in feeding experiments. The only explanation which can be offered at the present time for the inconsistent reports on the chemical nature of the visual purple is the extreme complexity and sensitiveness of the retinal tissue to heat and light.

There is every reason to believe that the colored lipid which forms a part of the visual purple may vary in chemical structure with the organism and that many kinds of visual purple may occur in nature. Perhaps the light-sensitive substance, which may be derived from a carotenoid and which may vary with the type of carotenoid taken as food, is modified by the tissue to correspond to the nature of the environment of the animal. Furthermore, it is probable that light-sensitive complex carotenoid derivatives occur in plant and nonretinal animal tissues.

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5. Brunner, O.; Baroni, E., and Kleinau, W.: Zur Kenntnis des Sehpurpurs, Ztschr. f. physiol. Chem. **236**:257, 1935.

6. von Euler, H., and Adler, E.: Visual Purple, Ark. Kemi. Min. Geol. **11B**:6, 1933; cited in Chem. Abstr. **28**:2048, 1934.

7. Wald, G.: Vitamin A in Eye Tissues, J. Gen. Physiol. **18**:905, 1935.

CONCLUSIONS

The visual purple is a lipoprotein composed of a protein conjugated with a golden yellow lipid having the properties of a carotenoid derivative There is a question whether or not the lipoprotein contains lecithin and cholesterol. The action of light on the visual purple liberates the golden yellow lipid, which, on further exposure to light, breaks down into a colorless noncarotenoid substance.

The analyses were performed with the technical assistance of Mr. W. Tauber.

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CHRONIC EDEMA OF THE CORNEA

REPORT OF A CASE

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The following case of unilateral edema of the cornea in a young adult of 30, unassociated with trauma, with ocular inflammation, infection or hypertension, or with any other apparent local or general disease, is reported because of the great rarity and the obscure nature and origin of the condition.

REPORT OF CASE

S. K., an intelligent and cooperative white man 30 years of age, a salesman, presented himself for examination at the clinic of the Brooklyn Eye and Ear Hospital, in the service of Dr. E. C. Place, on April 22, 1936. He complained of fogged vision of the right eye. The first visual disturbance was experienced in July 1932, while he was on a fishing trip. At that time vision became hazy but it cleared up entirely after a few hours of sleep. Hazy vision recurred at intervals up to December 1935. Since then the haziness had been present almost continuously. There was never any pain, irritation or sensation of a foreign body, redness of the eye, photophobia or lacrimation. There was no history of trauma. Rainbow halos around lights, of the type noted in glaucoma, were described. The visual impairment was more apparent on a cloudy day than a clear day, did not seem to change with the time of day, was not affected by worry or emotional upset and almost invariably appeared to be much less marked after the patient came out of the cinema.

The past history was not significant. The patient was married and had a normal child 5 years of age. He said he had never had a venereal disease. The general health had always been good; there were no gastro-intestinal complaints. There was no history of ocular or general disease in the family.

At the initial examination vision of the right eye was 20/40 and was unimproved with lenses; vision of the left eye was 20/15. The right eye presented normal adnexae; a white globe without any trace of conjunctival or pericorneal injection; dulness of the cornea, which on focal illumination with a Zeiss loupe was recognized as due to edema; a round pupil of the same size as its fellow and reacting actively to light, and an anterior chamber of normal depth. Corneal sensation, as tested with a fine wisp of cotton, was unimpaired. The tension, as measured with the Schiötz tonometer, was 19 mm. Slit lamp examination showed fine bedewing of the entire epithelium, which was most marked in the central area and similar to that observed in glaucoma; there were no deposits on the posterior corneal surface, nor was there any other evidence of inflammation. No staining with fluorescein was observed grossly or with the slit lamp. The left eye appeared to be normal in every respect, and slit lamp examination failed to show any corneal lesion similar to that in the right eye. With cycloplegia induced by homatropine hydrobromide retinoscopic examination revealed simple hypermetropia of low degree in

each eye. The red fundus reflex of the right eye with a plus 10 sphere was speckled, and the media other than the cornea and the fundus appeared normal in every respect. The peripheral and central visual fields and the blindspots showed no abnormalities.

Observed at intervals over a period of more than four months, the affected eye remained white and unirritated. Visual acuity fell to 20/70, corresponding to increasing corneal edema and loss of transparency. There never was any evidence of inflammation. There was no desquamation of epithelium. There was no corneal hypesthesia. The tension remained fairly constant at from 15 to 19 mm. (Schiötz), was uninfluenced to any appreciable extent by miotics or mydriatics, and showed little difference from that of the unaffected left eye. The lacrimal passages were permeable. There was no apparent lacrimal hyposecretion. Neither pupil dilated with the instillation of a 1:1,000 solution of epinephrine hydrochloride. On two occasions vision was found to be markedly improved after a stay of fifteen minutes in the dark room, once improving from 20/70 to 20/32, and the corneal cloudiness was observed grossly to have diminished. Atropine, pilocarpine, phenacaine, ethylmorphine hydrochloride and occlusion of the eye had no effect on the course of the edema.

On Sept. 2, 1936, the patient complained of pain, lacrimation and photophobia of the affected eye and presented on examination erosion of the epithelium and a large bulla of the lower portion of the epithelium. Vision was 20/100. The eye was anesthetized with butyn, and the entire epithelium was denuded to the limbus, the glistening Bowman's membrane being exposed. Vision now was 20/20. The fundus was clearly viewed and was normal in every respect. With the slit lamp three or four tiny grayish opacities were observed in the superficial portion of the stroma; there were no tears or folds in Descemet's membrane and no keratitis punctata, and the aqueous beam, which had on previous occasions been slightly clouded by the bedewed epithelium, was clear. Full strength tincture of iodine was applied to the entire surface of Bowman's membrane, and the eye was bandaged firmly. There followed a few months of recurrent erosions of the epithelium, which did not respond to the usual methods of therapy. The epithelium finally healed over but remained edematous, and the eye continued to be slightly painful and photophobic. The edema became gross and largely subepithelial, elevating the epithelium in the form of large bullae which were freely movable on pressure of the finger on the lower lid. The cornea at this stage was anesthetic. Vision was reduced to 10/200. On Feb. 4, 1937, the epithelium was again denuded, and a firm pressure bandage was applied. Microscopic examination showed sheets of well formed squamous epithelium; no keratinization was noted. The appearance of subepithelial edema persisted. Atropine sulfate, scopolamine hydrobromide, pilocarpine hydrochloride, phenacaine in solution of oxycyanide of mercury, pontocaine, thyroxin and cod liver oil were employed locally at various times, without benefit. A low fat (anticholesteremic), high calcium and vitamin-rich diet was given for several months, without apparent improvement.

On May 12 biomicroscopic examination by Dr. Milton Berliner revealed the following picture: With the finest beam the epithelium of the right eye was seen as a fine white line raised from Bowman's membrane by a dark area which may have contained fluid. This space (subepithelial) varied in depth, being deepest near the center of the cornea. There was a granular appearance of Bowman's membrane, which extended in places into the anterior third of the stroma. Definite folds of Descemet's membrane were seen, with many fine deposits of pigment on the posterior corneal surface. On retro-illumination the granular areas, seen arteriorly, took on the appearance of droplets (edema). On specular reflection

the folds were seen as dark lines over the endothelial cells. There were many crater-like depressions and fine dots, due to deposits in the posterior portion of the cornea. The appearance of the endothelium was similar to that seen in dystrophy. The corneal nerves were not enlarged; only a few were seen which were normal. The left eye was normal except for one or two annular superficial opacities resulting from old foreign bodies.

On May 17 the lids were sutured together. Ten weeks later the eye was perfectly white, painless and free from photophobia. The basic condition of edema persisted. There was a single, large, freely movable bulla that occupied the pupillary area, and the peripheral portion of the epithelium appeared finely bedewed.

A complete medical survey was made. General physical examination revealed a white man of 30 years, 5 feet, 5½ inches (166 cm.) in height and weighing 132 pounds (60 Kg.), without any gross physical defects. The blood pressure was 122 systolic and 80 diastolic. Examination of the nose and throat revealed a deviated nasal septum and residual tonsillar tags resulting from a previous operation; the paranasal sinuses transilluminated well. Roentgenograms of the sinuses showed no abnormality. Roentgenograms of the teeth disclosed an infected right lateral incisor tooth, which was extracted. Roentgenograms of the chest gave negative results. Urinalysis yielded negative findings. The Wassermann and Kahn tests of the blood were negative. Hematologic study showed 4,410,000 red cells, 82 per cent hemoglobin (Sahli), a color index of 0.93 and 12,000 white cells, with 61 per cent polymorphonuclear leukocytes, 31 per cent lymphocytes, 3 per cent eosinophils and 5 per cent monocytes. A later study of the blood showed 4 per cent eosinophils. The basal metabolic rate was + 2 per cent. The Mantoux test (carried out with from 0.1 to 1 mg. of old tuberculin) was negative. Study of the blood chemistry gave the following findings: a urea nitrogen content of 21.1 mg. per hundred cubic centimeters, a urea content of 45.1 mg., a sugar content of 95.5 mg., a calcium content of 9.6 mg., a phosphorus content of 4 mg. and a chloride content of 495 mg., an albumin content of 4.98 per cent and a globulin content of 2.31 per cent, a total cholesterol content of 333 mg. per hundred cubic centimeters in May 1936, of 222 mg. in June 1936 and of 310 mg. in February 1937, of which 85 mg. (or 27 per cent) was free. The dark adaptation of each eye when tested in September 1936 was normal, and that of the right eye when tested in March 1937 was again normal. On the latter date it was impossible to retest the affected eye. A thorough study with respect to allergy did not reveal any significant factor.

COMMENT

The essential feature in this case was edema of the cornea without any of the pathologic conditions which usually produce it. There was no associated gastro-intestinal or general disturbance. There was no urticaria. There was no history of trauma nor was there any sign of infection or inflammation. Under observation for four months the affected eye was white and unirritated, the epithelium did not desquamate, the corneal sensation was unimpaired and the ocular tension was not elevated. The symptoms of fogged vision and the rainbow colors around lights, as well as the appearance of bedewing of the corneal epithelium, suggested glaucoma, which was excluded by the normal tension, fundi and fields of vision. Recurrent erosions of the

epithelium were a complication. The edema progressed to a gross type, with the epithelial layer raised and forming large bullae. Biomicroscopic examination showed fluid under the epithelium and in the stroma, folds of Descemet's membrane and definite changes in the endothelium. The epithelium was entirely denuded on two occasions; after the first denudation tincture of iodine was applied, without benefit. Local therapy and dietary measures were likewise unavailing. Tarsorrhaphy was performed, which ameliorated the condition but did not remove the basic edema. A complete investigation, including consideration of endocrine imbalance, avitaminosis, allergy and a metabolic disorder as possible causative factors, did not yield positive results, with the exception of definite hypercholesteremia.

This condition falls into a class that forms a rather definite entity not previously described as such in the American or the British literature. Under "chronic edema of the cornea," "chronic intermittent corneal edema" and "chronic cyclic corneal edema" sixteen cases have been reported in the French and German literature. It occurs at any age from 10 to 70 years. Either sex may be affected. Heredity plays no rôle. One or both eyes may be affected. The symptoms are cloudy vision and rainbow colors around lights. There is no pain, photophobia, lacrimation or sensation of a foreign body; at most, there may be a sensation of fulness, roughness or warmth. The peculiar feature of the clouded vision is its variability: It may occur at intervals of days or weeks, with partial or complete subsidence, and there may be a regular daily cycle of greatest cloudiness on arising in the morning, which becomes less marked toward afternoon and subsides entirely toward evening. Not every patient presents this daily variation. There is no history of trauma. On examination there is no evidence of infection, inflammation, hypertension or any of the numerous possible causes of corneal edema. There is bedewing or vacuolation of the epithelium, which is most marked in the central area; the peripheral portion of the epithelium may be entirely normal. The corneal nerves may appear thickened and more clearly visible than in a normal cornea. Endothelial changes are usually demonstrable. Corneal sensation is not impaired. Desquamation of the epithelium is not a usual finding. Erosions of the epithelium, however, may occur. Elevation of the cholesterol content of the blood is a usual finding. The etiology is obscure, and there is no response to the usual methods of therapy.

Aubineau [1] first called attention to this entity in 1922 with a report of two cases, and he reported five additional cases in 1929. He credited

1. Aubineau, E.: Oedème cornéen et hypercholesterinémie, Bull. et mém. Soc. franç. d'opht. **35**:224, 1922; Ann. d'ocul. **159**:580, 1922; L'"oedème cornéen" des hypercholestérinémiques, Bull. et mém. Soc. franç. d'opht. **42**:283 (Aug.) 1929.

Despagnet [2] with describing the first case of this condition to be recorded. Löhlein [3] in 1913 described a case of fleeting corneal edema associated with urticaria; both phenomena were ascribed to the same unknown toxic cause. All Aubineau's patients had a history of gastro-intestinal or hepatic disturbance, and all showed hypercholesteremia. He ascribed the edema directly to the elevated cholesterol content, which he thought in some manner increased the power of imbibition of the cornea. With an anticholesteremic diet two patients were cured and the condition of one was ameliorated.

Marbaix [4] in 1932 reported a case of corneal edema of one eye associated with urticaria in a woman of 28 years. He found epithelial bedewing, clouding of the corneal parenchyma, thickening of the corneal nerves and endothelial changes. In addition, there was detachment of the endothelium in the same eye outside of the zone of edema and ascribed to injury sustained at birth. There was no response to diet or local therapy. He accepted the explanation of Aubineau and further suggested as a causative factor some toxic disturbance in the body resulting in both urticaria and corneal edema.

François [5] in 1933 reported in detail his observations in a case of corneal edema of the left eye in a man 52 years of age. There was no hypercholesteremia. There were a slightly elevated blood pressure and some renal involvement. Biomicroscopic examination revealed epithelial bedewing, clouding of the superficial and middle layers of the corneal stroma and thickening of the corneal nerves. He did not observe any changes in the deeper layers of the stroma, in Descemet's membrane or in the endothelium. He considered the condition to be a trophic disturbance and did a tarsorrhaphy, which resulted in complete restoration of the cornea to a normal status. The thickening of the corneal nerves suggested to him chronic neuritis of the cornea as the possible cause.

Hambresin [6] in 1934 reported a case of bilateral corneal edema in a man of 48 years who had been observed for a period of six years. There was slight hypercholesteremia. There were the characteristic epithelial bedewing and some alteration in the endothelium. An anti-cholesteremic diet had no effect. Sclerecto-iridectomy was performed on one eye, though the tension was not elevated, and the edema per-

2. Despagnet: Bull. Soc. d'opht. de Paris, 1888.

3. Löhlein: Selten Hornhautphänomen, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **39**:377, 1913; Rev. gén. d'opht. **33**:471, 1913.

4. Marbaix, M.: Un cas d'oedème de la cornée avec décollement probable de l'endothélium, Bull. Soc. belge d'opht., no. 64, April 1932, p. 117.

5. François, J.: L'oedème chronique de la cornée, Arch. d'opht. **50**:549 (Aug.) 1933.

6. Hambresin, L.: A propos d'un cas d'oedème cornée bilatéral, Bull. Soc. d'opht. de Paris, October 1934, p. 484.

sisted after the procedure. The edema seemed to disappear with the continued use of atropine sulfate and reappear with cessation of its use.

Haemmerli [7] in 1936 reported a case of typical chronic edema of the cornea, the patient being the youngest to be reported on, the onset dating back to the age of 9 or 10 years. Both corneas were affected. There was a definite cycle of cloudy vision. There were vacuoles in the epithelium, edema of the parenchyma and changes in the endothelium. All forms of therapy, both local and general, were unavailing. This author considered all the previous views as to the etiology of the condition untenable and the real cause unknown.

Blaauw,[8] in a clinical report of a case that he believed to belong to the group reported by Haemmerli, presented findings which were similar in many respects to those that I have recorded. There were recurrent erosions of the cornea, the formation of large freely movable bullae, marked clearing up of the edema after slit lamp examination in a dark room and lack of improvement after removal of the epithelium and the application of tincture of iodine. Cod liver oil locally applied seemed to have some salutary effect. Blaauw felt that the herpes virus played a rôle in the condition.

Graf [9] recently reported two cases with some interesting features. Each patient presented himself for examination at the very onset of the disturbance with a description of the typical daily cycle of varied vision, and in each the condition involved the left eye. In the first case, that of a photographer 32 years of age, there was a history of intermittent cramps in the thighs and legs, hepatic disturbance, slight hypercholesteremia and an eosinophil count of 9 per cent. Treatment with drugs, the roentgen rays and injections of insulin was of no benefit. The second patient, an optician aged 30, had a history of gonorrheal infection, excessive perspiration, hepatic disturbance, a normal cholesterol content of the blood and a positive Mantoux test. Autohemotherapy appeared to have some beneficial results. The condition was markedly improved after a period of observation of two years in the first case, and of one and a half years in the second case. The author remained skeptical as regards any form of therapy and stated that improvement occurred in spite of treatment. The principal observations on biomicroscopic examination were epithelial bedewing and vacuolation, changes in the size and visibility of the corneal nerves and endothelial changes. During the phase of greatest cloudiness of vision the epithelium pre-

7. Haemmerli, V.: Das zyklisch auftretende Hornhautödem, Klin. Monatsbl. f. Augenh. **97**:745 (Dec.) 1936.

8. Blaauw, E.: Ueber das zyklisch auftretende chronische Hornhautödem, Klin. Monatsbl. f. Augenh. **98**:665 (May) 1937.

9. Graf, K.: Chronisches intermittierendes Hornhautödem als Ausdruck einer Angioneurose, Ztschr. f. Augenh. **91**:278 (March) 1937.

sented vacuoles of varied sizes, the corneal nerves at the level of the deeper layers of the corneal parenchyma were easily discerned and much enlarged, and the endothelium showed defects, black spots, loss of contour and roughness of surface. In the phase of diminishing cloudiness of vision the epithelial vacuolation was reduced to bedewing and and then to normality, the corneal nerves became pale and thin and disappeared entirely, and the endothelium tended to return to a normal appearance in the early stages. A diet rich in cholesterol increased the corneal edema, and vice versa. If the disease persists for any length of time the original condition of the endothelium is never restored; eventually the borders of the cells become obliterated, and their normal form disappears. The entire posterior wall of the cornea becomes more homogeneous. Between the cells black spaces, which persist for months, appear. When the disease is not brought to a standstill the entire posterior third of the cornea becomes opaque, and then the picture of the endothelium becomes completely obliterated. In the second case very tiny, ill defined deposits (considered to be proliferations of the endothelium) resembling the precipitates of uveal inflammation were observed on the posterior surface of the cornea. Graf regarded the condition as vasomotor trophic neurosis, an ocular manifestation of angioneurotic edema. The transient edema associated with the unexplained cramps in the thighs and legs, excessive perspiration, hepatic and gastro-intestinal disturbance, urticaria and eosinophilia support this contention. The edema results from seepage of aqueous into the corneal substance after changes in the endothelial layer, which, when normal, forms a barrier to the penetration of fluid. The thickening of the corneal nerves is the result of imbibition of fluid through the nerve sheaths. The endothelial changes result from direct injury of angiotrophic influence or are secondary to altered chemical composition of the aqueous. That the condition is trophic follows from the fact that the central portion of the cornea, which is farthest removed from the source of nutrition, is predominantly affected.

The subject of chronic corneal edema has not escaped record in the American literature. Graves,[10] under the title "Epithelial Degenerations," discussed cases in which the patients presented as symptoms colored rings around lights and misty vision, did not have glaucoma and showed on biomicroscopic examination detectable epithelial changes. He particularly pointed out that such changes may be "transient, or remittent, or unobtrusive yet persistent." Gifford,[11] in a paper on "The Mild Form of Epithelial Dystrophy of the Cornea," mentioned the cases of Aubineau as

10. Graves, B., in Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, p. 476.

11. Gifford, S. R.: The Mild Form of Epithelial Dystrophy of the Cornea, Arch. Ophth. **7**:18 (Jan.) 1932.

apparently instances of the typical form of epithelial dystrophy, meaning Fuchs' dystrophy. In these cases of chronic corneal edema the clinical picture is definitely not that of Fuchs' dystrophy. In my opinion, these cases belong to the same group that Gifford described as cases of the mild form of epithelial dystrophy. Gifford specifically mentioned the typical daily cycle of variable vision, to which so much attention has been directed in the reports of cases of chronic corneal edema. The retention of normal corneal sensation and the absence of epithelial desquamation in cases in which the condition is uncomplicated, both of which are usual findings in chronic corneal edema, would seem to indicate that the epithelium is not intrinsically involved. There is little doubt that one is dealing with a primary disturbance of the endothelium, with secondary edema of the remainder of the cornea, in which the nerves partake. The undisturbed corneal sensation and the absence of pain militate against the conception of inflammation of the corneal nerves. The usual finding of hypercholesteremia is more than a coincidence and would seem to indicate a disturbance of metabolism. As in the case of all the corneal dystrophies, chronic corneal edema must be considered a nutritional or neurotrophic disturbance, but the basic etiology must remain for the present mere conjecture.

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AN INSTRUMENT FOR QUALITATIVE STUDY OF DARK ADAPTATION

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On several occasions I have presented data obtained by the examination of eyes for the study of dark adaptation.[1]

These tests were quantitative and required the instillation of a miotic which was occasionally objected to by the patient. Then, too, the procedure was a time-consuming one, since it took about one-half hour for the actual study to be made and a like amount of time for the plotting of a graph.

In an extensive study of unselected cases one expects to find a large number of normal persons with normal dark adaptation. It was therefore felt that much time could be saved if a simpler device was used which would quickly eliminate this group of normal persons. With such a test only a single reading of the minimum light visible is taken, with the light as the constant and time as the variable. This is in contrast to the test previously used, carried out with a more complicated instrument, in which the time was the constant and the light was the variable.

A rapid study of the light threshold is also of advantage, since the number of cases requested to be studied by the other clinics in the hospital has been steadily growing. This is probably due to the increasing interest in the subject as a result of the vast amount of contributions to the medical literature lately in which dysaptation [2] has been associated with vitamin A deficiency resulting from acute infections, particularly those of the nasal mucous membranes, from diseases of the skin and blood or from renal calculi, and also in which tests for dysaptation have been recommended for the detection of deficiency of vitamin A of children in the schools. One of the practical applications of this rapid test for hemeralopia cannot be overemphasized. It is possible to decrease the number of accidents occurring at night by acquainting persons suffering from hemeralopia with their affliction. The eyes of these persons.

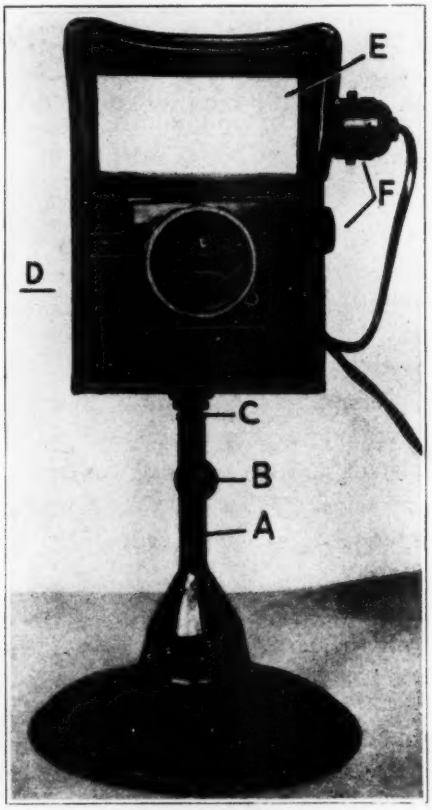
From the Laboratory of Ophthalmology, the Wills Hospital.

1. Feldman, Jacob B.: (*a*) Dark Adaptation as a Clinical Test, Arch. Ophth. **15**:1004 (June) 1936; (*b*) Dark Adaptation as a Clinical Test: Further Studies, ibid. **17**:648 (April) 1937.

2. Dysaptation is the term applied to dark adaptation in which time required for reaching the light threshold is prolonged beyond the normal period for a normal person, i. e., pathologic dark adaptation.

though they may be free from retinal pathologic changes, will indicate by a high light threshold as determined by the test that vitamin A deficiency is present.

The instrument for determining the light threshold (L. T.) [3] consists of a firm stand (*A*) in the tube of which is a thumb screw (*B*). The tube (*C*) of the lamp house (*D*) can be raised or lowered and



Instrument for the qualitative test of dark adaptation. *A* indicates the stand; *B*, the thumb screw; *C*, the tube of the lamp house; *D*, the dark adaptation chamber; *E*, the light adaptation chamber, and *F*, the switches. To use the instrument the light *E* is turned on for three minutes. This preexposure, which causes bleaching out of the visual purple, is then terminated, and the light in *D*, which illuminates the arrow, is turned on. In a normal eye the arrow should be seen and the direction which it takes should be recognized within a five minute period. Delay in seeing the arrow until five minutes or over classifies the condition as pathologic.

thus adjusted to suit the height of the particular patient and fixed in that position by the screw (*B*). In the lamp house (*D*) is a seasoned

3. Other terms used are light minimum (L. M.), minimum visible (M V.) and minimum light visible (M. L. V.).

light of daylight quality, constant in its voltage and illuminating the arrow or any other stimulus which it is desired to use. This arrow can be rotated at the will of the examiner without the knowledge of the patient. This helps to act as a check on the patient's veracity. Light adaptation is obtained with use of the lamp house (*E*). Switches (*F*) are used for illuminating the lamp houses for study of the light adaptation and the dark adaptation.

Since the saving of time is a factor, and as this study is only qualitative, no miotic to equalize the size of the pupils of every patient examined is necessary. A saving of from thirty to forty-five minutes is thus accomplished.

TECHNIC

1. The subject's vision is tested, and an ophthalmoscopic study is made. When the study of the dark adaptation is in the hands of a technician and the fundus cannot be examined, it may be assumed that in cases in which there is vision of 6/9 in each eye the maculae are intact.

2. All examinations for determination of the light threshold are made in a totally dark room.

3. The subject's forehead is placed against the frame of *E* for light adaptation for three minutes. A number of studies have been made as to the relative values of longer and shorter periods of light adaptation, as well as greater and lesser intensity of light in the light adaptation. The purpose of these studies was to obtain the effect which previous light adaptation had on subsequent readings for the light threshold.

The findings, however, brought out that a period of three minutes and a 100 watt light, used in my previous studies,[4] gave the most valid and reproducible results.

4. After three minutes the light is extinguished in the light adaptation lamp house (*E*) and switched on in the dark adaptation chamber (*D*), thus illuminating the arrow. The arrow is placed in the direction desired by the examiner.

The patient is moved back about 9 inches (23 cm.) from the arrow. Exact measurement of this distance is not necessary, as I have found that results have not been materially affected by slightly increasing the distance. The patient's hand is directed where the stimulus will be seen. This is done in order that the patient may not have to look in various wrong directions for the stimulus, with consequent delay in the time at which he actually sees the light.

Since time is the only important variable considered in this test, by indicating occasionally with the subject's hand where the light stimulus (the arrow) is to be seen the examiner fulfils a twofold purpose: 1. The patient is kept interested in the experiment. 2. Time is saved, and more valid results are obtained because the patient is looking in the right direction and can detect the light stimulus instantly. For a normal person the time that elapses from the termination of light adaptation to the recognition of the arrow and its position (the light threshold) should be under five minutes. A lapse of five minutes or over for reaching the light threshold is considered evidence of a pathologic condition, and the finding is grouped under dysaptation.

4. Feldman,[1a] p. 1006.

RESULTS OF EXAMINATION

One hundred and sixteen subjects were examined. Except for excluding patients with glaucoma,[5] these subjects were unselected.

Forty-seven subjects showed a high light threshold, i. e, pathologic dark adaptation, or dysaptation. Of these, thirty-one were females. An equal number of males and females showed the normal light threshold.

Since in this test both eyes are examined at one time, it should be noted that even fewer subjects than those considered to have a normal light threshold would be classed as having this if each eye was examined separately.

Obviously, if one eye has a pathologic condition and the other is normal, the arrow would be recognized by the good eye and the subject

TABLE 1.—*Persons Who Had a Normal Light Threshold and a Pathologic Condition of One or Both Eyes*

No. of Subjects	Condition
2	Chorioretinitis, with one eye affected
1	Exudative choroiditis, with one eye affected
1	Old choroiditis, with one eye affected
1*	Choroiditis, with both eyes affected
1	Choroiditis, with one eye affected
1	Old choroiditis, with both eyes affected
1	Bilateral macular choroiditis
1	Choked disk
1	Sclerosing keratitis

* I have noted several cases of choroiditis in which there was normal dark adaptation (footnote 1b, table 2, p. 651). It seems that the opinion of Traquar (Percival, A. S.: Notes on the Light Sense, Tr. Ophth. Soc. U. Kingdom **42**: 285, 1922) should be qualified to read that diffuse choroiditis tends to cause dysaptation more than choroiditis localized in a small area does. The small area of localized choroiditis may, unfortunately, not be included in the area examined, when the study of the light sense is made.

would be classed as having a normal light threshold. A test of the visual acuity and examination of the fundi of each patient would, however, acquaint the examiner with the ocular disorder present. In cases of sympathetic involvement [6] of an eye with disease of the other eye there may be a high or pathologic light threshold. This is the only type of case, to my knowledge, in which examination of an eye with an apparently normal fundus will show dysaptation, and the disorder might be misconstrued as vitamin A deficiency.[6]

CASES OF NORMAL LIGHT THRESHOLD

Included in the group of persons with a normal light threshold, it will be noted, are several in whom one or both eyes show a pathologic

5. The reason for excluding glaucoma is that in most cases of chronic glaucoma the light threshold is pathologic, which would greatly increase the number of cases of dysaptation.

6. Feldman,[1a] p. 1014.

condition, as listed in table 1. I have classed the subjects listed in the table and the rest of those with a normal light threshold as persons with normal regeneration of the visual purple and therefore as persons who had a normal amount of vitamin A, sufficient to permit such regeneration to take place. The least time in which the arrow was recognized by a person in this group was thirty seconds, and the greatest, up to but not including five minutes.

CASES OF PATHOLOGIC LIGHT THRESHOLD

Persons who took five minutes or more for the light stimulus to be recognized were considered to have a pathologic light threshold. All

TABLE 2.—*Persons Who Had a Pathologic Light Threshold and a Pathologic Condition*

No. of Subjects	Condition	Age, Years	Vision		Light Threshold Time
			Right Eye	Left Eye	
1	Hypertension—kidney disease	40	6/9	6/12	6 min.
1	Arteriosclerosis	65	6/9	6/9	7 min.
1	Old choroiditis of right eye	49	6/9	6/9	6 min.
1	Arteriosclerosis	69	6/15	6/15	5 min.
1	Choroidal retinitis; incipient cataract; hemorrhagic choroidal retinitis; corneal opacity	48	6/60	6/60	6 min.
1	High myopic; choroiditis	33	6/60	6/30	7 min., 30 sec.
1	Arteriosclerosis	39	6/6	6/6	12 min.
1	Renal calculi	34	6/6	6/6	10 min., 15 sec.
1	Recent undiagnosed acute infection	56	6/6	6/6	10 min., 30 sec.
1	Severe attack of grip 1 mo. previously	39	6/6	6/6	10 min., 45 sec.
1	Had 4 pelvic operations in last 2 yr	32	6/6	6/6	5 min.
1	Arthritis of knee	21	6/6	6/6	5 min.
1	Thyroid disease; basal metabolic rate +35 to +40	29	6/9	6/6	13 min.
1	Uses microscope	43	6/6	6/6	12 min.
1	Complains of photophobia	53	6/9	6/9	12 min., 30 sec.
1	Complains of difficulty in seeing at night	17	6/15	6/60	8 min., 15 sec.
1	Vague pain of right side—possibly due to renal calculi	40	6/5	6/15	8 min.
1	Hypertension and gastric ulcer	38	6/6	6/6	8 min., 15 sec.
1	Works where there is sun glare	27	6/6	6/6	5 min., 30 sec.
1	Diabetes and high blood pressure	50	6/6	6.6	8 min., 15 sec.
1	Asthma, sinusitis and chronic mastoiditis	51	6/6	6/6	10 min., 15 sec.
1	Sinus infection for 3 yr	10	6/6	6/6	10 min., 15 sec.
1	High myopia *	28	6/60	6/12	10 min., 30 sec.

* Attention has previously been called to this condition as a cause of dysaptation (footnote 1).

the subjects examined in this group had normal fundi unless otherwise stated. Table 2 gives this grouping.

The rest of the subjects who showed a light threshold higher than normal varied in age from 14 to 70 years and had irrelevant histories. Their light threshold time varied from five minutes to ten minutes and forty-five seconds; in many cases vision was as good as 6/5. These subjects could be classed as persons with failure of regeneration of the visual purple due to vitamin A deficiency, without subjective symptoms. Included in this group would also be those already mentioned in table 2 whose light threshold was analyzed who did not have a pathologic condition of the fundus.

CONCLUSIONS

An instrument for a rapid qualitative check on the light threshold is described and its method of use given. In this test the only variable is the time at which the light stimulus is first noted by the patient.

A period up to five minutes is considered a normal interval for reaching a normal light threshold.

The light threshold time ranged from thirty seconds (in a subject with a normal light threshold) to twelve and one-half minutes (in a subject with an abnormal light threshold).

One hundred and sixteen patients were examined, about 40 per cent of whom showed dysaptation, nearly 70 per cent of these being females.[7]

An equal number of males and females showed a normal light threshold.

Dr. William Ezickson aided in this study by checking some of the patients.

Physicians Building.

7. That females are more susceptible to dysaptation than males has been noted.[1a]

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

SURGICAL INTERVENTION FOR CATARACT

FROM THE PREOPERATIVE AND POSTOPERATIVE STANDPOINTS

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BALTIMORE

In this review I propose to take up the question of the survey of the case prior to operation for cataract, the choice of the operation to be performed and the most frequent postoperative complications and the way in which they may be combated.

SURVEY OF THE CASE

Of all the conditions which may mitigate against a successful outcome of an operation for cataract, that of a distant focus of infection is, in my opinion, undoubtedly the most frequent. Of such foci, that of diseased teeth is the one most frequently found. In the dental survey not only an inspection is desirable, but also a roentgen examination, since apical infections, abscesses and pyorrheal pockets are usually revealed by such a procedure. In case an apical abscess is revealed, extraction is the only satisfactory method of treatment, for although drainage and treatment may relieve the condition, the danger of a small area of infection remaining is too great to warrant subjecting the patient to this risk. When it is found necessary to extract teeth, because of either abscesses or pyorrheal conditions, sufficient time should elapse after this procedure before surgical intervention on the eye is attempted, in order to allow resolution to take place and the infection to be eliminated.

Steinbugler [1] questioned whether pyorrhea is ever a source of focal infection in ocular conditions, but if such a focus can cause arthritic conditions, certainly it could be a source of endogenous infection in the eye. The tonsils as a source of distant infection are not frequently at fault, although Jahnke [2] has seemingly shown conclusively a direct connection between septic tonsils and disease of the eye. At the age when cataracts are extracted, at least those of the senile type, the tonsils are usually atrophic (although this is not always the case), and therefore

1. Steinbugler, W. F. C.: New York State J. Med. **32**:1223, 1932.
2. Jahnke, W.: Ztschr. f. Augenh. **72**:354, 1930.

they should be investigated as a routine measure. A diseased gallbladder is also occasionally a focus, but in such cases, as in cases in which a diseased appendix is the focus (Gresser [3]), the patient is usually aware of his condition, and if the infection is serious he will seek relief before surgical intervention on the eye is considered.

Syphilis in the latent form as a cause of complications following extraction of cataract was discussed by Ballantyne,[4] who expressed the belief that patients should have a preliminary Wassermann test, and treatment, if necessary. Hardy [5] has reported a case in which syphilitic parenchymatous keratitis developed after extraction of a cataract in a patient with a latent syphilitic infection. While postoperative complications are more frequent in the presence of latent syphilis, healing may be just as rapid in a person with syphilis as in the normal person and free from all complications.

Diabetes is probably the outstanding constitutional disease that causes postoperative complications. While the danger from a general anesthetic in cases of diabetes is of little importance to the ophthalmic surgeon, the danger of postoperative infections and postoperative hemorrhages is greatly enhanced in such cases. Thus, Wheeler,[6] in a survey of 2,123 extractions of cataract performed at the New York Eye and Ear Infirmary, found that hemorrhages occurred in 28.94 per cent of the patients with glycosuria, while the average incidence was only 5 per cent in cases of uncomplicated cataract. According to Benedict,[7] however, the hemorrhagic tendency in patients with glycosuria is not benefited by the use of insulin but is increased by it. He advised discontinuance of insulin for at least four weeks prior to the operative procedure. Ballantyne [8] also noted that hemorrhages which occur in patients with diabetes are extremely slow in absorbing. Besides the tendency to hemorrhage, the diabetic patient shows a greater incidence of postoperative infections. Thus, Gifford [9] reported on a group of young diabetic patients 43 per cent of whom lost eyes from infections following extraction of cataract. According to Reese,[10] however, since the use of insulin the tendency to infection among diabetic patients is no greater than among nondiabetic patients. There is also a greater liability to postoperative iritis and iridocyclitis in the diabetic patient, but in spite of all these dangers it is often necessary to operate on diabetic patients.

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3. Gresser, E. B.: Am. J. Ophth. **14**:929, 1931.
4. Ballantyne, A. J.: (*a*) Tr. Ophth. Soc. U. Kingdom **52**:84, 1932; (*b*) **52**:86, 1932.
5. Hardy, W. F.: Am. J. Ophth. **5**:961, 1922.
6. Wheeler, J. M.: Tr. Am. Ophth. Soc. **14**:742, 1916.
7. Benedict, W. L.: Ohio State M. J. **21**:648, 1925.
8. Ballantyne.[4b]
9. Gifford, H.: Ophth. Rec. **20**:243, 1911.
10. Reese, F. M.: Internat. Clin. **3**:266, 1935.

The question of preparing the patient from the general standpoint should, of course, be left to a competent internist. It would seem from Benedict's experience that reduction of the sugar content of blood and urine to its minimum should be attempted by means of diet, but if the case is not controlled by these methods insulin should undoubtedly be used. However, the experience of my associates and me does not bear out that of Benedict's as to danger of postoperative hemorrhages after the use of insulin.

One cannot say that it is too dangerous to operate if the blood sugar content is over 200 mg. per hundred cubic centimeters and that it is safe to operate if the blood sugar content is under that amount, but the surgeon is driven almost by necessity to attempt surgical intervention if the patient is blind in both eyes and the condition is controlled as well as possible. In fact, Wilder [11] was so optimistic that he stated that complete control of the glycosuria is not essential and that the results of operation for cataract in the case of diabetic patients are as good as the results in the case of nondiabetic patients. This, however, is not the general opinion of operators of wide experience, who still feel that complications are more frequent in diabetic patients than in nondiabetic patients.

Hypertension as a complicating factor in cases of cataract cannot be ignored. Oláh [12] stated that even slight hypertension calls for venesection, with removal of from 200 to 250 cc. of blood on the day preceding the operation. Barrenechea [13] went even further and did a venesection on all patients over 50 years of age, unless there was some contraindication, removing from 250 to 300 cc. of blood. The reduction of the hypertension is, as a rule, the responsibility of the attending internist, and operative procedure is inaugurated only after he considers it safe. While it might be necessary to operate when the systolic pressure is above 200 mg. of mercury, the operation is done with a great deal of apprehension lest an intra-ocular or expulsive hemorrhage occur.

Nugent [14] in a large number of patients with cataract found the average systolic pressure to be 138, with a diastolic pressure of 80, and while, generally speaking, the patients with higher tensions were more likely to have complications, this was not necessarily so, since there seemed to be a greater number of complications in the group of patients with a systolic pressure of from 101 to 110 than in the group with a systolic pressure of from 141 to 150. While it seems hardly necessary

11. Wilder, R. M.: Symposium on Ocular Complications of Diabetes, Arch. Ophth. **12**:144 (July) 1934.

12. Oláh, Emil: Am. J. Ophth. **15**:626, 1932.

13. Barrenechea, A. S.: Arch. de oftal. hispano-am. **32**:207, 1932.

14. Nugent, O. B.: Tr. Sect. Ophth., A. M. A., 1933, p. 220.

to perform blood letting on every patient, as has been suggested, this is a procedure of merit in those cases in which the blood pressure is extremely high.

CHOICE OF OPERATION

Much has been written as to the operation to be selected for the different types of cataract, but there has been no absolute unanimity of opinion. Beach and McAdams [15] seem to be of the opinion that the intracapsular operation is applicable in cases of cataract of all types, while Knapp [16] agreed largely with Elschnig,[17] Kubik,[18] Wright [19] and others that the intracapsular operation is suitable in cases of senile cataract that is not in the intumescent stage and in cases of immature nuclear cataract. It is less suitable in cases of the morgagnian type and other hypermature types and in cases of cataract in the intumescent stage in which the capsule is so tense that grasping it is difficult. On the other hand, cataracts of the latter type can be extracted intracapsularly by using the eresiphake. The intracapsular type of operation is also considered unadapted in cases of cataract in patients under 50 years of age, because of the rather strong zonular fibers. Wright [20] decides against the intracapsular method in the presence of cough, asthma, high blood pressure, excitability and, in fact, practically any complicating general condition, as well as local rigidity of the pupil, high myopia, a fluid vitreous and glaucoma, and in the one-eyed patient. It would, therefore, seem that those who have had wide experience feel that extracapsular extraction is, on the whole, the safer procedure.

Choice of Operation for Congenital and Juvenile Cataracts.—In determining the type of operation to be performed on congenital cataracts, as well as on juvenile cataracts, the decision has to be made whether simple needling is to be performed or whether needling with subsequent lavage is to be practiced. The practice of needling combined with lavage after a few days is, in children, accompanied with such great danger of subsequent infection or injury from trauma that it seems to be rarely justified. The question of the time element is of little moment unless the child lives a considerable distance from a source of surgical help, so that the expense of travel becomes an important factor. What difference does it make to a child of 1 or 2 years if the period of absorption

15. Beach, S. J., and McAdams, W. R.: Intracapsular Extraction of Cataract in the Average Practice, Arch. Ophth. **15**:95 (Jan.) 1936.

16. Knapp, A.: Certain Aspects of the Intracapsular Extraction of Cataract by Forceps, Arch. Ophth. **16**:419 (Sept.) 1936.

17. Elschnig, E.: Die intrakapsulare Starextraktion, Berlin, Julius Springer, 1932.

18. Kubik, J.: Zentralbl. f. d. ges. Ophth. **32**:433, 1935.

19. Wright, R. E.: Am. J. Ophth. **16**:230, 1933.

20. Wright, R. E.: Am. J. Ophth. **20**:1, 1937.

is one month or six months? In a few cases the cataract may absorb with only one needling, although more than one is usually necessary, but a second operation is not necessary as long as the capsule is open and aqueous is coming in contact with the lenticular substance within the capsule. While Jackson [21] advocated the needling operation in patients up to 40 years of age, this is not the operation of choice unless slit lamp examination reveals that the lens is very soft and filled with large water clefts. In such cases the cataract may liquefy and absorb rather rapidly after the capsule is opened, but one should be ready at all times to open the anterior chamber and wash out the soft lenticular substance if secondary tension develops, which is not an unusual complication. Needling by the posterior route, which was recently advocated by Ballantyne [22] because of the lessened danger of swelling of the lens, does not seem to be well justified, since investigation has shown that the aqueous has the greater influence on the liquefying process which is the first essential to absorption.

ADVANTAGES AND DISADVANTAGES OF PRELIMINARY IRIDECTOMY

The question of the advisability of a preliminary iridectomy either as a routine measure or in cases of exceptional or complicated conditions, such as immature cataract, is one that has caused considerable discussion and called forth much difference of opinion. It is thought that von Graefe was the first to practice this procedure. Some operators since then have practiced it as a routine procedure, but a greater number employ it only in cases of more or less exceptional or unusual conditions. The chief advantages seem to be the following: first, the ripening effect on immature cataracts; second, the fact that at the time of the subsequent extraction there is no hemorrhage into the anterior chamber from the cut iris; third, the fact that one of the most painful parts of the operation does not occur at the time of the extraction, and, fourth, the fact that the surgeon becomes somewhat acquainted with the reaction of the patient to surgical procedure. Of the more or less doubtful advantages claimed by some might be mentioned the fact that there are an unobstructed region for insertion of the cystitome or capsule forceps, absence of iritis due to pressure and less need for a subsequent needling operation.

Of the disadvantages, the following seem to be of great moment: first, two penetrations of the ocular coats, with the accompanying dangers of exogenous infection; second, the increased expense of two hospitalizations, with the accompanying disturbance of home routine, and, third, the increased apprehension of the patient (it has been my experience that

21. Jackson, E.: Arch. Ophth. **42**:596, 1913.
22. Ballantyne, A. J.: Brit. J. Ophth. **20**:540, 1936.

when a patient undergoes a second operation he seems to dread it more than the first operation and complains of more pain than at the time of the initial operation).

In regard to the more recent methods of operating, the question of exogenous infection remains practically the same as in former times. The problem of lessening the pain is of less moment, for with the use of more prolonged application of the local anesthetic and especially with the use of retrobulbar injections, the pain caused by the operation is no longer an important factor. While there is undoubtedly a limited field for preliminary iridectomy, it is becoming progressively less important, especially as an operation to hasten ripening, since the intracapsular method of extraction is advocated for the immature cataract. My experience agrees with that of Fuchs, who stated that he rarely found indications for the operation.

POSTOPERATIVE COMPLICATIONS

Owing to the multiplicity of postoperative complications, it will be impossible to discuss all of them in a review of this scope, but the more frequent ones will be dealt with briefly.

Glaucoma.—Glaucoma following extraction of a cataract may result from the fact that the iris or the capsule of the lens has prolapsed and healed into the wound, which condition seems to disturb the normal drainage sufficiently to cause increased tension. On the other hand, there may be an ingrowth of epithelium into the anterior chamber, which, by completely epitheliating the filtration angle greatly or entirely, reduces filtration, with a resulting increase in intra-ocular pressure. I have observed two cases of this type in which the diagnosis was confirmed by microscopic studies, a third case in which a clinical diagnosis was made by means of the slit lamp and a fourth in which the condition was discovered in an enucleated eye. In the two cases in which the diagnosis was definite the eye was operated on by filtration methods, but even repeated operations failed to reduce the tension permanently. Vail [23] reported a case in which a clinical diagnosis of downgrowth of epithelium into the anterior chamber was made; the condition seemed to be benefited by radiation, but the diagnosis was not confirmed by microscopic investigation, so there is a possibility that this diagnosis was incorrect. In one of my cases, which has previously been mentioned, in which the eye was subsequently enucleated and the diagnosis was confirmed, the condition was treated by radiation, without any apparent beneficial result. If the increased tension is due to incarcerated iris or capsule, freeing the wound from the adherent capsule or iris will at times reduce the tension to normal. If the condition is due to epithelial

23. Vail, D. T., Jr.: Tr. Am. Ophth. Soc. **33**:306, 1935.

ingrowth, a filtering operation should be attempted, as well as the use of radiation, but so far the results of such treatment have not been encouraging.

Endophthalmitis Phaco-Anaphylactica.—The rather pronounced reactions following extraction of cataract in a few cases, especially in those in which there is considerable soft lenticular substance, has been noted for many years. Verhoeff and Lemoine advocated the term endophthalmitis phaco-anaphylactica for this condition. A spirited discussion took place as to whether this condition is due to an allergic reaction, to some type of infection or even to the irritating properties of the lens itself. Gifford,[24] in this country, and Rötth,[25] as well as Riehm,[26] abroad, were on the negative side. Burky,[27] however, was able to produce a similar condition experimentally in rabbits by first sensitizing them to lens protein plus staphylococcus toxin, after which needling of the lens produced a marked ocular reaction. If the injections of the same combination were continued, the rabbit became desensitized, at which time needling of the lens produced little, if any, reaction. On the basis of these observations Burky and Henton[28] treated two patients whose condition was clinically diagnosed as endophthalmitis phaco-anaphylactica, with rapid and satisfactory improvement. It would therefore seem that if this complication arose, the combined treatment of lens protein with staphylococcus toxin was the method of handling such conditions.

Sympathetic Ophthalmia.—One of the most disappointing and disastrous complications to develop after extraction of cataract is sympathetic ophthalmia. Fortunately, this condition is infrequent, the incidence ranging from 0.027 to 0.5 per cent. However, if, in a case of bilateral cataract, after successful removal has been accomplished in one eye and after what seemed like a satisfactory extraction has been carried out on the opposite eye, it is found in the course of a few weeks that the eye previously operated on is inflamed and that there are photophobia, deposits on Descemet's membrane and all the other signs of sympathetic inflammation, the condition is almost certain to be sympathetic ophthalmia. Theobald[29] found that the condition occurred only twice in 7,444 cases in which extraction of cataract was performed, but in these cases the reaction was violent and ultimate vision very poor.

The question of treatment in cases of sympathetic ophthalmia is difficult because of the uncertain etiology. The use of large doses of

24. Gifford, S.: Tr. Sect. Ophth., A. M. A., 1925, p. 82.
25. Rötth, A.: Arch. Ophth. **55**:103, 1926.
26. Riehm, W.: Klin. Monatsbl. f. Augenh. **88**:62, 1932.
27. Burky, E.: Experimental Endophthalmitis Phaco-Anaphylactica in Rabbits. Arch. Ophth. **12**:536 (Oct.) 1934.
28. Burky, E. L., and Henton, H. C.: Am. J. Ophth. **19**:782, 1936.
29. Theobald, G.: Am. J. Ophth. **12**:597, 1930.

salicylates, as advocated by Gifford, has seemed, in my hands, to be as efficacious as any other treatment. Arsenicals given intravenously are sometimes useful, and Verhoeff [30] expressed the opinion that diphtheria antitoxin in large doses is effective. Desensitization to uveal pigment was recommended by Woods,[31] while Friedenwald [32] stated the opinion that ultraviolet irradiation of the general cutaneous surface is of benefit. Unfortunately, the sympathetic ophthalmia which occurs after surgical intervention for cataract seems to be violent, and treatment seems in most cases to be disappointing.

Postoperative Hemorrhage.—Postoperative hemorrhage into the anterior chamber is frequent, especially in diabetic patients, as was shown by Wheeler.[6] Vail [33] found, on averaging available figures, that it occurred in 7.59 per cent of all cases in which extraction of cataract was performed. Since it is more frequent in those cases in which iridectomy has been performed, it seems safe to assume that it frequently comes from the iris, but it is also seen occasionally in cases in which simple extraction has been done. The corneoconjunctival incision may be the source of the hemorrhage in a certain number of cases.

It has been suggested that the use of epinephrine at the time of operation was a factor in the production of the hyphemia, but Jensen,[34] who studied the question, came to the conclusion that there was no material difference in the frequency of bleeding in those cases in which epinephrine was used and in those in which it was not used. Schneider [35] noted a severe hemorrhage into the anterior chamber in a case of hemophilia, which caused a rupture of the wound that required suturing. The blood eventually absorbed, leaving an obstructing membrane. Prior to operation on the other eye, the patient was given intramuscular injections of calcium for three days before the operation, and a hypodermic injection of morphine hydrochloride and scopolamine hydrobromide one hour before the operation. This eye showed no bleeding after the operation, healed without complications and had good resulting vision. While hemorrhage into the anterior chamber is comparatively frequent and usually not serious, this cannot be said of expulsive hemorrhage, which is rare, fortunately (the incidence being 1 case in 1,000), but which practically always destroys the eye. This type of bleeding may occur at the conclusion of the incision or may be delayed as long as the tenth day after the operation.

30. Verhoeff, F.: Tr. Am. Ophth. Soc. **24**:173, 1926.
31. Woods, A.: Am. J. Ophth. **9**:52, 1926.
32. Friedenwald, J.: Am. J. Ophth. **17**:1008, 1934.
33. Vail, D. T., Jr.: Tr. Am. Ophth. Soc. **31**:496, 1933.
34. Jensen, V. A.: Acta ophth. **10**:382, 1932
35. Schneider, R.: Klin. Monatsbl. f. Augenh. **91**:365, 1933.

Most of the hemorrhages from the choroidal vessels which cause loss of the eye are not of the expulsive type that necessitate immediate enucleation but are often discovered at one of the dressings after the patient has complained of some restlessness and pain in the eye. Treatment of the milder conditions of hyphemia consist of the use of atropine and heat locally combined with the use of calcium internally. If repeated bleeding takes place, solution of brain extract may be given hypodermically, as recommended by Black,[36] who reported a case in which the hemorrhages ceased after its use. If the hemorrhage is one of the more severe types, injection of a preparation of ergot, solution of posterior pituitary (10 minims) and epinephrine hydrochloride (10 minims) given two or three times a day, as recommended by Ziegler,[37] may be used as a prophylactic measure, and may also be used if bleeding is actually taking place. Ziegler also advocated a hypodermic injection of morphine, hyoscine hydrobromide and pilocarpine hydrochloride. He stated that in spite of all treatment such eyes are usually lost. Thilliez,[38] on the other hand, claimed that the hemorrhage is immediately arrested after the subcutaneous injection of epinephrine hydrochloride and ergot. Although this opinion seems somewhat overenthusiastic, it behooves the surgeon to keep in mind these recommendations in case of an emergency. There seems to be a marked difference of opinion as to whether or not procaine hydrochloride and epinephrine hydrochloride, injected retrobulbarly, have a tendency to increase the number of cases of intraocular bleeding.

Inflammations, Purulent and Nonpurulent.—Infections of the eye following operations for cataract may be exogenous or endogenous, the former being the most frequent. Since greater care has been exercised in the preparation of the instruments and the conjunctival sac, as well as the patient, the incidence of postoperative infections has greatly diminished (from 5.47 to 1.5 per cent, according to Collins [39]).

The nonpurulent inflammations may be the result either of an infection or of trauma, the latter being often the cause. Ballantyne [8] advocated the use of atropine and hot compresses locally, with large doses of salicylates and sodium bicarbonate combined with mercurial inunctions. Foreign proteins are also helpful in many cases.

The treatment of the purulent infections is usually disappointing, but not every attempt is a failure; therefore, one should combat the

36. Black, M.: Am. J. Ophth. **7**:539, 1924.

37. Ziegler, S. L., in Crisp, W. H., and Finnoff, W. C.: Contributions to Ophthalmic Science, Dedicated to Dr. Edward Jackson in Honor of His Seventieth Birthday by His Pupils and Colleagues in the United States, Menasha, Wis., George Banta Publishing Company, 1926, p. 7.

38. Thilliez: Bull. Soc. franç. d'opht. **35**:406, 1922.

39. Collins, T.: Tr. Ophth. Soc. U. Kingdom **34**:18, 1914.

condition as actively as possible. If one can obtain a culture showing the chief offending organism, a more logical treatment can be outlined. Prior to the identifying of the organism, the use of atropine with a local antiseptic such as a 20 per cent solution of mild protein silver or a 0.1 per cent solution of acriflavine, combined with the use of hot moist compresses applied almost continuously, is to be recommended. If the organism is a streptococcus, prontosil,[39a] given intramuscularly, may be tried. If the organism is a pneumococcus, ethylhydrocupreine hydrochloride in a 2 to 5 per cent solution may be helpful. The use of foreign protein in this condition often aids materially. Hartlieb [40] obtained good results by the intravenous use of 10 cc. of a 2 per cent solution of acriflavine hydrochloride and the intramuscular administration of 2 cc. of a mixture of partial antigens containing the metabolic products of various bacteria, lipoids from bile and neutral fats. Stargardt [41] expressed the belief that all soft tissue at the edges of the wound should be curetted away and the anterior chamber washed out with either physiologic solution of sodium chloride or a 1 : 5,000 solution of mercury oxycyanide. Butler [42] as well as Ellett,[43] advised cauterizing the margin of the wound with the actual cautery. If resolution does take place it is always a slow process.

Detachment of the Retina and Choroid.—Though detachment of the retina has been known since 1868, it has until recently been regarded as a rather infrequent complication in cases of extraction of cataract, although Fuchs [44] in 1900 found an incidence of 4.7 per cent. Recently O'Brien [45] stated that it occurred in 93 per cent of consecutive cases in which extraction of cataract was performed and in which the eye was examined immediately after the extraction. He reported that in this type of detachment the retina always becomes reattached, with no impairment of vision. Occasionally the detachment appears after several months and often in cases in which there is a history of a greater or less jar. In this group of patients there is usually the picture of typical detached retina, with loss of transparency. At times a rent is found in the detached portion, while frequently no such lesion can be detected. The prognosis is usually poor in these cases, but Cruise [46] reported

39a. Prontosil is a derivative of sulfanilamide. The preparation used is said to be the disodium salt of 4'-sulfamidophenyl-2-azo-7-acetylamino-1-hydroxynaphthalene-3, 6-disulfonic acid.

40. Hartlieb, R.: Klin. Monatsbl. f. Augenh. **93**:526, 1934.

41. Stargardt, K.: Ztschr. f. Augenh. **43**:321, 1920.

42. Butler, T. Harrison: Tr. Ophth. Soc. U. Kingdom **40**:181, 1920.

43. Ellett, E.: J. Ophth., Otol. & Laryng. **10**:227, 1916.

44. Fuchs, E.: Arch. f. Ophth. **51**:199, 1900.

45. O'Brien, C. S.: Arch. Ophth. **16**:655 (Oct.) 1936.

46. Cruise, R.: Detachment Following Simple Extraction of Cataract, Arch. Ophth. **15**:576 (March) 1936.

successful results after the Šafář operation, and Hine [47] reported a cure by the same method in a case in which detachment occurred after the needling operation. Shapland [48] reported 30 cases of detachment following cataract, in 40 per cent of which holes were found. Of the patients who were subjected to operation, 18 per cent were cured. While the results in the hands of most operators have not been good in cases of this type of detachment, there is no doubt but that an attempt should be made to reattach the retina by one of the usual methods, the method employed being that which has seemed most successful in the hands of the attending surgeon.

Postoperative Delirium.—While postoperative delirium is a troublesome complication, it is usually not serious, since uncovering of the eye not operated on is usually all that is necessary to restore mental equilibrium. However, if the patient does not have a special nurse he may disturb the dressing or get out of bed and cause damage to the eye in the form of prolapse of the iris or an intra-ocular hemorrhage. Greenwood [49] found that the condition occurred in from 2.5 to 3 per cent of all cases in which extraction of cataract was performed. According to my own experience this seems somewhat too high, but possibly slight mental confusion might be present as frequently as Greenwood's figures indicate. McMullen [50] expressed the opinion that the mental distress which patients on whom extraction of cataract has been performed frequently have is due to anxiety as to whether they are going to see again. Bruns [51] allowed a group of patients on whom he operated to go home after the operation; he observed no mental disturbance in these cases, but it would seem that this treatment is slightly too heroic. The use of morphine and scopolamine or some of the milder hypnotics is usually all that is necessary to control the condition.

47. Hine, M. L.: Am. J. Ophth. **18**:558, 1935.

48. Shapland, C. D.: Am. J. Ophth. **17**:1159, 1934.

49. Greenwood, A.: Mental Disturbances Following Operations for Cataract, Tr. Sect. Ophth., A. M. A. 1928, p. 233.

50. McMullen, W. H.: Brit. J. Ophth. **20**:657, 1936.

51. Bruns, H. D.: Tr. Am. Ophth. Soc. **14**:473, 1916.

News and Notes

EDITED BY DR. JOHN HERBERT WAITE

GENERAL NEWS

Publication of "Confinia neurologica."—A new international journal, to be called *Confinia neurologica,* is now being published by von S. Karger, A. G., Basel, Switzerland. The journal will be edited by Dr. E. Spiegel, of the Temple University School of Medicine. As far as the borderland of neurology is concerned, the new journal will endeavor to maintain the mutual relationship of neurology and surgery, otolaryngology and ophthalmology, syphilology, endocrinology and radiology. Complete and preliminary original articles, reviewing articles, society transactions and book reviews will be published. Articles may appear in English, French or German; the original papers will have a summary in all three languages. The summaries will be translated by the journal staff, if the author so desires.

SOCIETY NEWS

Eye Section of the Philadelphia County Medical Society.—The following papers were read at the scientific meeting of the Eye Section of the Philadelphia County Medical Society, held Oct. 7, 1937:

"Pathology of Sympathetic Ophthalmia," by Dr. Bernard Samuels, with discussion by Dr. Perce DeLong.

"Methods and Value of Examining Conjunctival Secretions," by Dr. Edgar B. Burchell, with discussion by Dr. Wilfred E. Fry.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Bacteriology and Serology

THEORETICAL BASIS FOR THE USE OF IMMUNOGEN B IN IMMUNIZATION OF THE EYE. G. G. BURSUK, Vestnik oftal. **10**: 500, 1937.

Bursuk has been doing experimental work with antivirus since 1928. He observed that the products of activity of microbes accumulate in the antivirus and come in contact with the leukocytes which rush to the site of immunization. As a result of this interrelationship between the antivirus and the leukocytic ferments a new substance formed, which causes a sensitive reaction to the infection, i. e., rapid mobilization of the cellular elements and rapid formation of antibodies, the index of immunity of the tissues. This substance which appears during the process of immunization Bursuk named immunogen B. Through various experiments material accumulated which characterized the immunizing properties of immunogen B against the staphylococcus. The agglutination reaction was taken as the most accurate and simple. The same verified strain of Staphylococcus aureus has always been used for the agglutination. Bursuk also examined the change in the leukocyte count of the animals after the injection of immunogen B. Nine tables and one drawing demonstrate the results of the experimental study of immunization of the eye with immunogen B.

The following conclusions are drawn:

1. The immunogen against the staphylococcus causes a marked increase in the index of agglutination.

2. It also causes marked leukocytosis.

3. The increase in agglutinins and in leukocytes depends on the number of injections and the dose.

4. The action of immunogen B is specific, as it causes an increase in agglutinins for staphylococci only.

5. This was particularly marked in those animals the eyes of which were infected by staphylococci.

6. The concentration of the agglutinins and of the leukocytes was more marked near the site of the immunization; hence the first injection of the immunogen should be introduced at the site of the infection.

7. Retrobulbar injections are preferred, as they do not irritate the eye and cause increase in antibodies in the media; antibodies were prevalent in the aqueous of the posterior chamber twenty-four hours after the first injection of immunogen B, as compared with their number in blood vessels remote from the site of immunization.

O. SITCHEVSKA.

Conjunctiva

OCULAR PEMPHIGUS. W. J. KAPUSCINSKI, Ann. d'ocul. **174**: 451 (July) 1937.

Ocular pemphigus usually follows pemphigus of the skin and usually begins at the same time as pemphigus of other mucosa, particularly buccal and pharyngeal. However it sometimes occurs as an apparently primary ocular disorder.

Four cases of ocular pemphigus are detailed and are of interest because of the complete clinical picture. Kapuscinski concludes that ocular pemphigus is a disease characterized by primary degeneration of the mucosa of the conjunctiva, especially the epithelium. The pathologic processes in the cornea are limited to the superficial layer and may be similar to the changes that take place in the conjunctiva. This chronic inflammatory condition is characterized by cellular infiltration and is purely a secondary phenomenon which is caused by the invasion by microbes of tissue already pathologically altered by degeneration due to pemphigus.

The article has fifteen illustrative figures and a bibliography.

S. H. MCKEE.

SYPHILITIC CONJUNCTIVITIS. A. ŠPANIĆ, Arch. f. Ophth. **137**: 312 (June) 1937.

Severe, subacute, unilateral, chiefly infiltrating conjunctivitis associated with ipsilateral interstitial keratitis occurred in a syphilitic youth 18 years of age after the primary genital lesion. The conjunctivitis resembled trachoma but remained unchanged under antitrachomatous treatment. Pathologically, diffuse infiltration (by lymphocytes, plasma cells and polymorphonuclears), with a few epithelioids and giant cells, was seen. Under antisyphilitic treatment the disease subsided promptly.

P. C. KRONFELD.

ETIOLOGY OF GONOBLENNORRHEA NEONATORUM: REPORT OF A CASE. E. JOHANSSON, Klin. Monatsbl. f. Augenh. **97**: 775 (Dec.) 1936.

Three days after birth an infant had rhinitis, with a watery discharge, which became purulent a few days later, when difficulty of nasal breathing developed and traces of blood appeared in the nasal discharge. On the fourteenth day the lids were slightly swollen, and a conjunctival discharge was noted. Gonoblennorrhea neonatorum with chemosis was evident in each eye on the sixteenth day. Gonococci were present in the conjunctival and nasal discharge. Solution of silver nitrate had been properly instilled after the method of Kredé. This case suggests the necessity of considering not only diphtheria and syphilis, but also gonorrhea, as a cause of rhinitis in the new-born. Even stomatitis gonorrhoeica has been observed, due, probably, to gonorrheal conjunctivitis. Johanssen suggests following Kredé's treatment with the use of less irritating preparations in every case of maternal gonorrheal vaginitis, so as to avoid late infections of the type described.

K. L. STOLL.

Cornea and Sclera

CYCLIC EDEMA OF THE CORNEA: REPORT OF A CASE. V. HAEMMERLI, Klin. Monatsbl. f. Augenh. **97:** 745 (Dec.) 1936.

A laborer aged 19, employed at a quarry, had noticed in his ninth and tenth years that his sight was less acute in the morning than in the afternoon, and this condition had become worse in recent years; at first transient, it grew permanent finally. He never experienced headache, vomiting or epiphora, nor did he see colored halos around lights. The family history furnished no information as to ocular disease of the parents, sister and brother.

The patient's eyes were examined nearly every hour during the day and night; they appeared practically normal at 4 p. m. Early in the morning the entire cornea of each eye looked "finely dewed," probably as a result of fine epithelial changes. After 4 a. m. the fine "dewy" spots were more numerous and were scattered over all the layers of the cornea which now appeared thicker under the slit lamp. Incidentally, the intra-ocular tension was higher and fine "vacuoles" were visible in the epithelium of the cornea a few hours later. The vacuoles were less evident about 9 a. m. and had disappeared at about 11 a. m. A few green spots were caused by fluorescein while the vacuoles presented.

Despagnet and François described edema of the cornea unaccompanied by irritation or increased tension. Including Haemmerli's case, twelve cases of cyclic edema of the cornea have been described, the age of the patients ranging between 19 and 60 years. Haemmerli's patient was the youngest (aged 10 at the onset of the disturbance). The cycle occurred within twenty-four hours in every patient. Some authors considered hypercholesteremia, others, trophic disturbances, as the etiologic factor. The etiology is unsettled, in Haemmerli's opinion. Mydriatics, miotics, epinephrine and a cholesterol-poor diet have been used, and sclerectomy and iridectomy have been done, without result, by divers authors.

K. L. STOLL.

CORNEAL TRANSPLANTS TAKEN FROM CADAVERS: REPORT OF CASES. Z. NIŽETIĆ, Klin. Monatsbl. f. Augenh. **97:** 756 (Dec.) 1936.

Nižetić discusses divers technical questions relative to corneal transplantation and then reports his results in thirty-nine operations of this kind. In twenty-four of these cases the corneal transplants were taken from eyes of patients who had died of various causes, including enteritis, bronchopneumonia, asphyxia, intoxication with sodium hydrate, and accidents. The corneal disks were removed within from four to twenty-four hours after death. The ages of the deceased varied between 11 months and 30 years; eighteen of them were children. The technic is described. No enucleation was performed. No transplant was lost, but all of them healed, even two in cases in which infection occurred; these, however, grew opaque later on. Among these twenty-four cases, in five the transplant remained clear, in one case for over three years; in six it remained transparent, and in thirteen it became opaque. These results resemble those of other authors. Nižetić thinks that permanent transparency is less dependent on the material than on the substratum chosen for transplantation. The more the normal corneal elements are preserved in the substratum, the better will be the transparency of the

transplant. The cases were not especially selected with reference to fitness for a favorable keratoplasty, because the author's purpose was the study of the material; only a few of these eyes were free from complicated total leukoma.

K. L. STOLL.

TWO CASES OF PERMANENTLY CLEAR HEALING CORNEAL TRANSPLANTS IN OPTICAL KERATOPLASTY. S. OCHI, Klin. Monatsbl. f. Augenh. **97:** 761 (Dec.) 1936.

Ochi reports two cases in which homoplastic, circumscribed, penetrating optical keratoplasty was performed. The patients were a woman aged 22 and a man of the same age. The transplants were taken from eyes enucleated for glioma in children who were 3 years and 4 years and 8 months old, respectively. Ochi selects for implantation the least vascularized portion of the cornea. He considers the transplants taken from children the best. The blood group of the patient and the donor need not be the same. The result, in Ochi's opinion, depends more on the corneal changes of the patient's eye than on the selection of the spot for the transplantation. It depends, secondly, on the age of the donor and finally on the technical experience of the operator.

K. L. STOLL.

Experimental Pathology

IRITIS PRODUCED IN RABBITS' EYES BY THE INTRAVENOUS INJECTION OF CRUDE AND PURIFIED CULTURES OF BACTERIA ISOLATED FROM PATIENTS WITH CERTAIN INFLAMMATORY EYE DISEASES. C. BERENS, E. L. NILSON and G. H. CHAPMAN, Am. J. Ophth. **19:** 1060 (Dec.) 1936.

The authors give the following summary and conclusions:

"Iritis was produced in rabbits by the intravenous injection of either primary or purified cultures from 19 of 21 patients with acute or chronic eye diseases, and in 11 of 14 controls (laboratory assistants, healthy children and patients with arthritis and thyrotoxicosis).

"Positive results were obtained with various microörganisms as follows: streptococci (alpha, beta, and gamma types), staphylococci (albus and aureus), colon bacilli, nonlactose fermenters, enterococci, and Friedländer bacilli. Of the 51 primary cultures from patients with eye disease, 25.5 percent produced iritis in rabbits and 39 percent caused death of the rabbits before examination or too early for the production of eye symptoms. Of the 35 primary cultures from the control group, 26 percent produced iritis and 60 percent caused death of the rabbits before iritis was observed. The high mortality of the rabbits injected with primary nasal cultures accounts for the large number of undetermined results.

"Iritis was produced by 44 percent of 61 purified strains of streptococci from patients with eye disease as compared with 29 percent of 69 strains from persons in the control group.

"Of the other organisms from patients with eye disease, 36 percent of the 22 purified strains of staphylococci, members of the colon group, and enterococci produced iritis. The results were undetermined in 18

percent. In the control group, 41 percent of the strains of staphylococci, members of the colon group, and Friedländer bacilli produced iritis. The results were undetermined in 25 per cent.

"Of the total of 134 cultures from patients with eye disease, 36 percent produced iritis while 17.9 percent were undetermined. Of the total of 116 cultures from persons in the control group, 29.2 percent produced iritis in rabbits, while 21.5 percent were undetermined.

"Toxicity, as measured by in-vitro tests, did not seem to be related to the iritis-producing power of streptococci and staphylococci. Seventy percent of the organisms which produced iritis gave positive toxicity reactions, whereas 60 percent of the strains which did not produce iritis also gave positive toxicity reactions.

"It is concluded that, while iritis is produced in rabbit's eyes by various cultures of bacteria, this property is not characteristic of any one bacterial genus, neither is it distinctly a property of cultures from patients with inflammatory eye diseases."

W. S. REESE.

General

THE DEGREE AND PREVALENCE OF VITAMIN A DEFICIENCY IN ADULTS. H. JEGHERS, J. A. M. A. **109:** 756 (Sept. 4) 1937.

The article deals with the study of one hundred and sixty-two students of the Boston University School of Medicine with especial regard to dark adaptation.

The author's summary and conclusions are as follows:

Vitamin A deficiency is common in adults and varies from a photometrically detectable phase to the complete clinical syndrome. In the group of medical students, 35 per cent had low photometer readings, and 12 per cent had clinical manifestations of the deficiency. The chief manifestations, in the order of their frequency, were night blindness, photophobia, dry skin, dry conjunctivae, blepharitis and follicular hyperkeratosis. The factors producing the deficiency were analyzed, and showed that the skipping of meals and poor choice of foods were chiefly responsible. After dietary analyses it was concluded that 4,000 U. S. P. units of vitamin A daily represents the minimal requirement for a healthy adult. Infections were more numerous and severe among the students with vitamin A deficiency than among the other students. Further evidence that it is dangerous for the hemeralope to drive an automobile at night was obtained. Photometric evidence of night blindness appeared in six days, and subjective evidence in five weeks, after the production of pure vitamin A deficiency in an experimental subject. Night blindness preceded gross epithelial changes.

W. ZENTMAYER.

OCULAR HEADACHE. D. S. STEWART, Brit. M. J. **2:** 59 (July 10) 1937.

Headache is a symptom of speculative etiology. A patient consults his physician only if the headache is persistent, recurrent or disabling. In cases of chronic headache it is as necessary to examine the optic disks as to test the urine, the blood pressure or the knee jerk. One should also determine if the patient can read 6/6 (that is, the seventh

line of the usual Snellen test type from a distance of 6 meters) with each eye separately. This test eliminates uncorrected but not overcorrected myopia.

Any severe illness, operation or accident, if it occurs in the late thirties or after, may leave the accommodation permanently lessened.

The normal relation between accommodation and convergence, and the imbalance between these two, which may occur in cases of uncorrected hyperopia are well known. In children the imbalance may lead to strabismus; in adults, asthenopia or headache may result.

Stewart thinks that the binocular balance at the reading distance is important in ruling out this group of conditions. He recommends the use of the Maddox convergiometer for discovering these disorders. Correction of refractive errors often restores the normal muscle balance for near.

In conclusion, he states that examination of the visual acuity, the optic disks and the lateral muscle balance at the reading distance should indicate which patients need ophthalmic attention.

W. F. DUGGAN.

OCULAR PAIN: ITS TREATMENT BY ANESTHESIA OF THE SPHENOPALATINE GANGLION AND ORBITAL ALCOHOLIZATION. A. MAGITOT, Ann. d'ocul. **174:** 361 (June) 1937.

Ocular pain brings the patient to the ophthalmologist as frequently as visual disturbances. Eliminating such causes as swelling of the lids, burns, and conditions caused by infection of a neighboring sinus or dental abscess, Magitot discusses only those related to the eyeball itself. Among these may be mentioned corneal erosions, which can usually be helped by the instillation of an analgesic.

Certain diseases of the anterior segment of the eye, such as keratoconjunctivitis, scleritis, parenchymatous keratitis and iritis, are usually accompanied by considerable pain, which is treated by certain drugs, the choice of which varies for individual patients, and by the local application of hot fomentations. One may also try a method which is without risk and which sometimes gives remarkable relief; that is the subconjunctival injection of air. The technic is simple and consists in provoking subconjunctival emphysema. Magitot has been using this method since 1912 and injects the air at four cardinal points, obtaining a large swelling about the cornea. The emphysema is usually absorbed in about twenty-four hours but may remain a little longer. Sometimes these methods are insufficient, and it is necessary to use others. He suggests anesthesia of the sphenopalatine ganglion and injections of alcohol into the orbit. These methods are described in detail, and a commentary on the results is added.

S. H. MCKEE.

General Diseases

FIBROCYSTIC DISEASE OF THE FRONTAL BONE (PAGET'S OSTEITIS). R. E. WRIGHT, Brit. J. Ophth. **21:** 364 (July) 1937.

A Hindu girl aged 20 showed a soft cystic tumor projecting from beneath the left supra-orbital rim and proptosis of the left eye. A roentgengram revealed a "nigger wool" skull.

An incision was made into the cyst through the brow; a portion of the frontal bone was removed; the cyst was drained from the temporal side, and the skin was allowed to fall in. The periosteum at the orbital rim was continuous with the membranous wall or capsule of the cyst, which intervened between the orbital contents and the cyst cavity.

The histopathologic observations were consistent with the view that this was an instance of localized fibrocystic disease of bone falling within the group in which there is no change in the calcium and the phosphate metabolism.

The article is illustrated.

W. ZENTMAYER.

Glaucoma

RESULTS OF THE SURGERY OF GLAUCOMA. L. BOTHMAN and M. J. BLAESS, Am. J. Ophth. **19:** 1072 (Dec.) 1936.

This article is an analysis of 100 cases (143 eyes) of primary glaucoma, including 7 in which there were retinal hemorrhages before operation and 8 (13 eyes) in which there was hydrophthalmos. No case in which there was evidence of iridocyclitis is included. The results of various operations on these eyes are discussed and tabulated. The authors give the following summary and conclusions:

"The anterior chamber was shallow and the cornea steamy when the tension was more than 50 mm. Hg.

"The 'middle pressure' was approximately the same as the intraocular tension in glaucomatous eyes under miotics and closely approximated that figure in patients with less than 50 mm. of pressure.

"Fields constricted to within a few degrees of the fixation point in glaucomatous eyes are no contraindication to surgical treatment.

"One late infection in fistulating operations occurred among 156 eyes and this one recovered 0.6 — 3 vision.

"The Elliot trephining operation was found to be the most satisfactory surgical procedure in chronic glaucoma."

W. S. REESE.

GLAUCOMA IN AMBLYOPIA. S. V. ABRAHAM, Am. J. Ophth. **19:** 1094 (Dec.) 1936.

Abraham believes that glaucoma occurs infrequently in amblyopic eyes, as the blood supply is decreased in cases of subnormally functioning organs, and therefore these cannot call on the same extensive vascular response that is obtained in normal tissue. He believes that the reports in the literature bear out this hypothesis. He reports a case of acute glaucoma in an eye undoubtedly amblyopic following early convergent strabismus, and states the following conclusions:

"1. A case of acute glaucoma in an amblyopic eye is presented. 2. It is suggested that subnormally functioning eyes tend to be less susceptible to primary glaucoma. This seems to be true for amblyopic eyes. 3. Relief from 'eye strain,' especially as it occurs in hyperopia, is particularly desirable in glaucomatous patients. 4. The relation of a disturbed inflow to the etiology of glaucoma is emphasized."

W. S. REESE.

Lacrimal Apparatus

ACUTE DACRYOADENITIS DUE TO THE MORAX-AXENFELD DIPLOBACILLUS. R. E. WRIGHT, R. BAHADUR and K. KOMAN NAYAR, Brit. J. Ophth. **21**:367 (July) 1937.

A Hindu youth aged 19 had, in addition to inflammation of the right lacrimal gland, enlargement and tenderness of the right preauricular gland. Pulp from the preauricular gland yielded a pure culture of the diplobacillus of Morax and Axenfeld.

W. ZENTMAYER.

LACRIMAL ELIMINATION OF DEXTROSE IN DIABETES. D. MICHAIL, P. VANCEA and N. ZOLOG, Compt. rend. Soc. de biol. **125:** 194, 1937.

Normally the tears contain no detectable dextrose, but in the diabetic patients examined the dextrose content ranged from 32 to 84 mg. per hundred cubic centimeters. It is of interest that the lacrimal gland is thus concerned in the elimination of products of abnormal metabolism.

J. E. LEBENSOHN.

DIVERTICULUM OF THE LACRIMAL SAC WITH SPECIAL REGARD TO ROENTGENOLOGIC FINDINGS. F. SPINELLI, Arch. di ottal. **44:** 89 (Feb.) 1937.

The literature on the subject of diverticulum of the lacrimal sac is reviewed, and a case of this condition is reported. The patient, a woman of 31, had noticed a swelling over the right lacrimal sac since the age of 8 years. It increased in size gradually, being the size of a small nut when seen by Spinelli. It extended to the middle of the lower lid and was soft. Pressure on the tumor caused no escape of pus through the canaliculi, but a few drops of pus could be expressed by pressure on the region of the lacrimal sac. Irrigation into the nose was possible. Puncture of the cyst revealed pus containing diplococci. Its contents were replaced by 20 per cent iodized oil. Roentgenograms showed a large oval shadow without apparent connection with the sac. Later roentgenograms, however, made after injecting the lacrimal sac with iodized oil, showed that the cyst was filled from the sac through a small communication.

The lacrimal sac and the cyst were removed. Sections showed the cyst to be composed of tissue identical with that of the lacrimal sac, a septum of similar tissue separating the two cavities. Apparently the septum was arranged so as to form a valve which opened only on pressure from the side of the lacrimal sac, in which respect the condition seems to the author unique among those reported in the literature.

S. R. GIFFORD.

Lens

PNEUMATIC EXTRACTION OF THE LENS BY ZONULAR RUPTURE. H. LAGRANGE, Ann. d'ocul. **174:** 387 (June) 1937.

Lagrange has manufactured a pneumatic extractor for the extraction of cataract, which he wishes to bring before practitioners and which

substitutes for other methods rupture of the zonule by traction at its ciliary insertion.

The method of anesthesia is explained, and the necessity of care in following out the procedure is emphasized. The avoidance of haste is most necessary. The contraindications are also given.

The article does not lend itself to abstraction. There are five pages of illustrations showing the details of the method and the appliances used.

S. H. McKee.

Physicochemical Influences Affecting Opacification of the Lens. P. Reiss, J. Nordmann and C. Reiss, Compt. rend. Soc. de biol. **125:** 464, 1937.

To a filtrate of triturated lenses a small amount of an oxidizing agent, such as hydrogen peroxide, quinone or 2, 6-dichlorphenolindophenol, was added. Preserved by a drop of toluene, the mixture was then incubated at 37 C. for twenty-four hours. Only a slight clouding was noted in solutions of normal or alkaline p_H, but even a slight acidification, as at p_H 6.5, produced a marked effect. With the oxidation-reduction potential of the solution with the added hydrogen peroxide at + 368 millivolts, opacity is manifest at p_H 6.9. Fresh lenses immersed in these solutions reacted in the same manner.

J. E. Lebensohn.

Neurology

Ocular Changes in Multiple Sclerosis. D. Marshall and R. G. Laird, Am. J. Ophth. **19:** 1085 (Dec.) 1936.

Marshall and Laird emphasize the frequency and early appearance of ocular changes in multiple sclerosis. They report a series of one hundred cases in which this condition was diagnosed by the department of neurology of the University of Michigan Medical School; 75 per cent of the patients had a routine examination by the department of ophthalmology. The ocular manifestations of multiple sclerosis are discussed, and a case of multiple sclerosis in which ocular changes were found is reported. The following summary is given:

"A review of history and ocular findings in 100 consecutive cases of multiple sclerosis is presented. In general the frequency of ocular pathology corresponds closely with statistics reported by previous writers. In addition a case of hemianopsia is reported in which diagnosis of multiple sclerosis was based entirely on field changes. The article aims to remind ophthalmologists and neurologists of the important and frequent part the eye plays in the diagnosis of the disease."

W. S. Reese.

Suprasellar Meningioma: Removal in Toto, with Prompt and Lasting Amelioration of Vision. P. Puech, P. Halbron and L. Guillaumat, Bull. Soc. d'opht. de Paris, February 1937, p. 71.

A patient aged 28 years had rapid loss of vision, which decreased to 5/10 for the right eye and to 2/10 for the left eye. There were pale disks and bitemporal hemianopia. The results of neurologic exam-

ination were negative. There was slight involvement of the clinoid processes, although the sella turcica was normal. A diagnosis of suprasellar tumor was made. The visual fields are reproduced, and six drawings of the large tumor made during operation are included in the article. Differentiation from adenoma or craniopharyngioma was easily made. One month after operation the visual fields were normal, and vision of the right eye was 9/10 and that of the left eye 10/10. The tumor was removed in a single mass.

L. L. MAYER.

NYSTAGMUS IN TUMORS OF THORACIC PORTION OF SPINAL MEDULLA. H. HOFF and O. PÖTZL, Med. Klin. **33:** 598 (April 30) 1937.

Hoff and Pötzl observed spontaneous nystagmus in three of six patients with tumors in the thoracic region of the spinal medulla. They admit that because of the small number of cases the figures are of no significance, but they think that the characteristics of the nystagmus which appeared in these cases deserve attention. They observed that it had a slow and a rapid component. The rapid component was in the direction of the visual movements; it was entirely horizontal and appeared especially when the patient was standing, sitting or in the abdominal or the dorsal position. Turning the pelvis or rotating the legs arrested the nystagmus. These seemed to be the only movements that caused its cessation. To be sure, in one case completion of the lateral recumbent position was necessary to arrest it. After surgical removal of the tumor from the thoracic portion of the spinal medulla, the nystagmus disappeared completely, or only slight traces of it remained. As long as the nystagmus was at the peak of its development pelvic movements toward either side and each lateral recumbent position had the same effect, in spite of the fact that in the first two cases the spastic and paretic conditions were not equally severe on the two sides. In evaluating the significance of the described observations the authors point out that these indicate the need of careful examination of the nystagmus that develops in cases of tumor of the spinal cord. Even if spontaneous nystagmus is absent, it is advisable to subject the patients to the same examination as that carried out on patients with spontaneous nystagmus in order to determine whether some change in the position might not perhaps elicit latent nystagmus. The significance of this type of nystagmus is slight in regard to the differential diagnosis, because it occurs also in rare cases of multiple sclerosis; but it should be impressed on the nonspecialist in this field that the existence of nystagmus does not necessarily speak either against a tumor of the thoracic portion of the spinal cord or for multiple sclerosis.

J. A. M. A. (W. ZENTMAYER.)

Ocular Muscles

LESIONS OF THE OCULAR MUSCLES BY DIRECT TRAUMA. L. VERICO, Arch. di ottal. **44:** 65 (Feb.) 1937.

Two cases of lesion of an ocular muscle or of ocular muscles caused by trauma are reported. In the first case a wound was caused by a piece

of iron, which tore the bulbar conjunctiva, severed the internal rectus muscle near its insertion and partially severed the reflected portion of the superior oblique muscle. Operative repair was performed immediately. The severed portion of the internal rectus muscle with Tenon's capsule was united to the stump of the tendon, and portions of the tendon of the superior oblique muscle which presented in the wound were removed. Fairly good motility was obtained, but a divergence of 10 degrees was left, for which tenotomy of the external rectus muscle was performed only two weeks later. The eye became straight in the primary position, and there was slight limitation of motion only in the lower inner field.

The second patient received a severe laceration of the lids by falling on the handle of a door. The laceration exposed the lower half of the globe and severed the inferior rectus muscle at the junction of its tendinous and muscular portions. Immediate suture was performed. The muscle was found by grasping deeply into the orbit and determining whether the muscle tissue was held by the forceps by traction produced on attempted movements of the eye. The tissue was sutured to the stump of the tendon, and an excellent result was obtained. After a month motility was practically normal, and no diplopia was present.

The mechanism of injuries to the ocular muscles and the relative value of early and of late operative intervention for such injuries are discussed. A bibliography is given.

S. R. GIFFORD.

Ocular Muscles

INFLUENCE OF LUMBAR PUNCTURE ON THE PARALYSIS OR PARESIS OF THE EXTRINSIC OCULAR MUSCLES. F. CARAMAZZA, Rev. otoneuro-oftal. **12:** 529 (Nov.-Dec.) 1936.

Caramazza describes nine cases of paralysis of ocular muscles of differing etiology. There was no increase in the intracranial pressure. In six cases improvement followed lumbar puncture. Results can be obtained only when the paralysis is of recent origin. On the basis of his observations the author considers the subject worthy of study by ophthalmologists.

G. BONACCOLTO.

Operations

TARSORRHAPHIA MEDIALIS VERA. N. I. SHIMKIN, Brit. J. Ophth. **21:** 343 (July) 1937.

The author's summary in part supplies an adequate abstract of the article.

Shimkin offers a new method of operation, tarsorrhaphia medialis vera, for ectropion paralyticum of the lower lid in cases in which the punctum lacrimale and the canaliculus lacrimalis are so occluded that the physiologic function of the lower lacrimal passage cannot be restored.

The operation consists in drawing the inner part of the lower lid, extending from the punctum lacrimale up to the inner canthus, by means of two loop sutures upward and inside a deep pocket in the inner part of the upper lid; the pocket is made by splitting the margin of the upper lid along the intermarginal line into two plates, extending from the punctum lacrimale superior up to the ligamentum internum.

The punctum and canaliculus of the upper lid remain intact in this operation.

On the medial part of the lower lid the skin is previously excised, and the conjunctiva and margin of the lid are scarified, which causes adhesion, when healing, to the tissues of the walls of the pocket in the upper lid. All the three layers are fastened together with a single knot suture in the newly formed canthus of the eye and with a loop stitch inward from it.

The operation is called tarsorrhaphia medialis vera, as the tarsus of each lid is sutured in the inner canthus of the eye, and not only the skin of the lids, as in all the methods of tarsorrhaphia medialis devised up until now. This operation makes it possible for the surgeon to gage the necessary distance to raise and shorten the lower lid. The cosmetic result is satisfactory.

W. ZENTMAYER.

ANESTHESIA INDUCED BY SODIUM EVIPAL IN OCULAR SURGERY. Z. NIZETIC, Ann. d'ocul. **174:** 375 (June) 1937.

The choice of an anesthetic depends not always on the physician, but often on the patient who says he will have the operation only if he is "put to sleep first." This is usually true of operations on the globe.

Nizetic describes the use of evipal (C = C = cyclohexenyl = N = methylbarbituric acid) and gives a table of data for 115 cases in which this anesthetic was used, showing the sex and age of the patients, the kind of operation, the amount of drug used and the results. He summarizes the findings as follows:

Narcosis induced by sodium evipal is, without doubt, like that induced by other narcotics, a serious matter, but the author has not had the slightest accident or serious complication from the use of this drug, probably because he kept the dose moderate. The results have been most satisfactory in ocular surgery, in which this form of anesthesia has such particular advantages as allowing freedom of the operative field, improvement in securing asepsis, and absence of preoperative and postoperative inconveniences. Sodium evipal is a satisfactory drug for inducing anesthesia and may be used in urgent ocular procedures and in major operations, and the length of anesthesia is sufficient for all ocular operations (?—S. H. M.). In operation for detachment of the retina Nizetic believes sodium evipal to be the anesthetic of choice.

S. H. MCKEE.

Orbit, Eyeball and Accessory Sinuses

THE INFLAMMATORY PSEUDOTUMORS OF THE ORBIT (BIRCH-HIRSCHFELD). A. FRANCESCHETTI and E. RUTISHAUSER, Arch. f. Ophth. **137:** 93 (April) 1937.

The term inflammatory pseudotumor of the orbit (Birch-Hirschfeld) designates a number of diseases which clinically cannot be distinguished from neoplasms and which only on pathologic examination prove to be inflammatory in nature. In this paper four cases of such a pseudotumor are reported; in three of the cases the process had started in the lacrimal gland, and in the fourth case it originated from the

periapical granuloma of a premolar. The pathologic picture was that of diffuse chronic inflammation in the case of the growth presumably of dental origin. In the other three cases the pseudotumor consisted chiefly of follicles in which, in two of the three cases, tubercle bacilli could be demonstrated histologically. In one of these two cases the bacilli proved to be pathogenic for the guinea-pig. In view of the relative frequency of pseudotumor the authors recommend that a biopsy be carried out early in all cases of suspected orbital tumor. Partial excision of a pseudotumor often leads to complete cure but may also cause considerable limitation of the movements of the eye and lids. The authors therefore favor simple incision instead of partial excision. "The formation of lymph follicles is a typical reaction of the orbital tissues."

P. C. KRONFELD.

CEPHALOCELE ORBITAE ANTERIOR. A. MIKLÓS, Arch. f. Ophth. **137:** 222 (June) 1937.

Miklós reports two cases of anterior cephalocele of the orbit (one case is that of a patient operated on by Kreiker and reported by him in 1922). Exploratory puncture is recommended to establish the diagnosis. Early operation is indicated unless the cephalocele is accompanied by more serious developmental defects or by hydrocephalus.

P. C. KRONFELD.

Physiology

AN INVESTIGATION INTO THE THEORIES ON THE FORMATION AND EXIT OF THE INTRA-OCULAR FLUIDS. J. D. ROBERTSON, Brit. J. Ophth. **21:** 401 (Aug.) 1937.

This study is considered under the following heads: historical, experimental, discussion, conclusions and summary. The paper occupies forty-five pages of the issue. Under the heading discussion there is an exhaustive analysis of the experimental work of other investigators. Robertson's conclusions and summary are as follows:

Conclusions.—The views put forward in favor of dialysis for the production of the intra-ocular fluids have been carefully investigated, and it is suggested that the evidence is unconvincing.

There is ample evidence to show that in the production of the aqueous there must be an expenditure of energy in the posterior chamber, for the membrane through which fluid passes into the chamber to become aqueous has an irreversible permeability.

There is evidence to show that there is no chemical equilibrium between the blood and the aqueous. Easily diffusible constituents, such as urea, sugar and uric acid, are not present in equal concentrations in blood as compared with the corresponding aqueous.

There is evidence that a physical equilibrium does not exist between the blood and the aqueous and that the equilibrium level of the intra-ocular pressure is not maintained by the hydrostatic force in the capillaries minus the difference in the osmotic pressure between the aqueous and the blood.

It is sug[illegible] that the evidence, chemical, hydrostatic and osmotic, points to the [illegible]mation of the aqueous by a process of secretion, and

in addition there is evidence of the necessity for expenditure of energy in its formation.

Summary.—It is held that the formation of aqueous takes place by a process of secretion at the ciliary body and that this fluid moves forward to the angle of the anterior chamber and is actively absorbed into the canal of Schlemm by some process other than osmosis.

W. ZENTMAYER.

FURTHER RESEARCH ON RESPIRATION OF OCULAR TISSUES. D. MICHAIL and P. VANCEA, Compt. rend. Soc. de biol. **125:** 185, 1937.

Experiments both in vitro and in vivo indicate that epinephrine, insulin, cevitamic acid and the roentgen rays stimulate the oxidizing activity of lenticular and corneal tissue and tissue of the lacrimal gland. This investigation was suggested by a study of naphthalene cataract, in which the oxidizing power of the lens was found to be reduced from the onset of intoxication, preceding the retinal and lenticular lesions by several days and reaching its maximum with complete cataractous development.

J. E. LEBENSOHN.

HYDROGEN ION CONCENTRATION OF THE TEARS. A. GARDILČIĆ, Arch. f. Ophth. **137:** 71 (April) 1937.

In a case of cicatricial ectropion with exposure of parts of the upper fornix, the spontaneously formed lacrimal fluid was aspirated directly from the outlets of the lacrimal gland. The rate at which fluid appeared on the conjunctival surface is not given in the paper. The entire conjunctiva was in a state of chronic inflammation. The p_H of the undiluted lacrimal fluid (determined with a quinhydrone electrode and a capillary electrometer) was found to vary between 7.53 and 7.95. Dilution with double-distilled water lowered the p_H. The secretion of the conjunctiva is more alkaline than the tears.

P. C. KRONFELD.

Retina and Optic Nerve

ANEURYSM OF THE INTERNAL CAROTID ARTERY WITH ATROPHY AND COMPRESSION OF THE OPTIC NERVE. J. O. WETZEL, Am. J. Ophth. **19:** 1053 (Dec.) 1936.

Wetzel briefly reviews the literature on aneurysm of the internal carotid artery producing changes in the optic nerve due to pressure. He reports a case of this disorder in a 39 year old housewife who showed a secondary atrophy of the optic nerve of the left eye, which progressed. The patient was observed for one year, at the end of which she died suddenly. Autopsy revealed extensive hemorrhage of the brain and a much flattened and stretched left optic nerve over the surface of an aneurysm of the left internal carotid artery. Other changes were observed in the body. The author discusses the etiology of aneurysm.

W. S. REESE.

MARKED AMELIORATION OF TABETIC OPTIC ATROPHY BY TREATMENT WITH AN ARSENIC AND BISMUTH PREPARATION. H. LAGRANGE and P. LEFÈVRE, Bull. Soc. d'opht. de Paris, February 1937, p. 58.

Lagrange was prompted by the report of Bégué, who found atrophy of the optic nerve following the use of acetarsone, to ask the specialist in syphilis, Lefèvre, to report on a certain patient. The patient had paralysis of the third nerve, associated with ptosis and diplopia. Treatment with mercurials injected intravenously resulted in cure of these paralyses. Prolonged treatment with intramuscular injections of acetarsone in doses of 1 Gm. resulted in decreased visual acuity after the third injection. Visual acuity was reduced from 9/10 for the right eye and from 10/10 for the left eye to 7/10 for each eye. Acetarsone was discontinued. Bismuth and mercury preparations were given, but vision decreased, and atrophy of the optic nerve was noted. The reduction of vision was to 2/10 for the right eye and to 1/50 for the left eye. A preparation of arsenic and bismuth in combination was then given. Visual acuity increased to 7/10 for the right eye and to 3/10 for the left eye. The authors feel that a trivalent arsenical is indicated in cases in which atrophy of the optic nerve is present.

L. L. MAYER.

PAPILLITIS AND STELLATE RETINITIS OF THE RIGHT EYE; DISCUSSION OF THE ETIOLOGY. DUBOIS-POULSEN and R. ROSSANO, Bull. Soc. d'opht. de Paris, February 1937, p. 99.

In the examination of a woman aged 36 the authors noted papillitis of the right eye and reduction of vision to 1/10; on second examination, five days later, a star figure in the macula, was seen. A thorough and painstaking general examination failed to reveal the cause of the condition. There was no evidence of kidney disease, or of hypertension, either local or general. This condition has not infrequently been reported in the literature (Bull, Eales, Leber, Larsson, Moore, Miles, Miley, Webster and Schlesinger having described cases) as evidence of focal infection of some distant organ. The tests used in the present case are reported in detail. In addition to the question of the diseases previously mentioned, the authors discuss that of toxic disease, disease of blood cells, trauma, syphilis and tuberculosis. Therapy directed specifically toward these disease processes was of no avail in the authors' case. It is necessary to conclude that the etiologic factor is unknown, and the authors ask for further discussion to reveal the cause of such a syndrome.

L. L. MAYER.

CEREBRAL ANGIOSPASM: SIGNIFICANCE OF RETINAL ARTERIAL HYPERTENSION. F. SCHOUSBOÉ, Bull. Soc. d'opht. de Paris, February 1937, p. 112.

From a study of fifty-three patients with clinical characteristics of cerebral lesions of angiospastic origin the following conclusions may be drawn:

1. Forty-three per cent of the patients had no modification of the retinal circulation (local spasm).

2. Fifty-seven per cent had accompanying retinal arterial hypertension.

In 79 per cent of the latter the condition was transitory; in 34 per cent of this group spasm of retinal arteries was bilateral, and in 66 per cent the condition was on the same side as the cerebral lesion. In thirty-three patients the etiologic factors could be determined, and these were grouped as follows:

Posttraumatic epilepsy	1
Essential hypertension	12
Syphilitic hypertension	5
Hypertension with endogenous intoxication (urea)	8
Hypertension with exogenous intoxication (tobacco or alcohol)	3
Raynaud's disease	1
Reflex angiospasm	3

L. L. MAYER.

NEW ANATOMIC FINDINGS IN CHOKED DISK. C. BEHR, Arch. f. Ophth. **137**: 1 (April) 1937.

Behr condemns alcohol-hardened specimens of choked disk and the conclusions based on such specimens, on the grounds that the rapid dehydration of the edematous nerve is bound to lead to the formation of holes in the tissues which were not present intra vitam. For seven years he has, therefore, embedded his specimens of choked disk in gelatin. Sections prepared in this manner reveal no gaps between the bundles of nerve fibers and the septums, no empty spaces under the pia and no "loosening" of the axial strand. The essential pathologic change visible in these gelatin sections is edema of certain portions of the nerve fiber substance itself. In cross-sections this edema manifests itself as multiple but minute empty spaces within the bundles of fibers. The multiplicity of these spaces gives the affected portion a cystic appearance. In early stages of choked disk only the subpial bundles show this appearance, but with the progress and the longer duration of the disease bundles of fibers situated more axially become affected. The extent of the pathologic process parallels the loss of the visual field. In longitudinal sections the bundles of fibers show swelling except at the places of contact with the septums. Thus a "rosary" shape of the bundles results. The septums and the axial strand appear compressed and not edematous. The entire extracranial portion of the optic nerve shows this change. The swelling is confined to the disk and does not involve the adjoining retina.

Ophthalmoscopically, choked disk starts as a circumscribed swelling of one quadrant of the disk. According to Behr, this fact can be explained only by his own theory, namely, that of the existence of a centripetal intrafascicular lymph stream in the optic nerve, which is interfered with by increased intracranial pressure. Only live nerve fiber tissue is capable of undergoing the edematous change which underlies the "choking," another fact which is most easily explained by Behr's theory. The hydrops of the intervaginal spaces cannot exert pressure on the nerve because, according to Behr, these spaces communicate with the orbit.

P. C. KRONFELD.

VITREOUS AND RETINAL DETACHMENT. K. LINDNER, Arch. f. Ophth. **137:** 157 (June) 1937.

The paper deals chiefly with the theory of Leber, Gonin and Lindner that retinal tears are caused by the pulling effect of an abnormal vitreous. Leber formulated his views as follows: A disease of the peripheral choroid causes shrinking and, consequently, detachment of the vitreous. Progression of this shrinking process puts the retina under tension and produces the retinal tear. Gonin's addition to this theory was the assumption that the tear occurs at the place where previous retinochoroidal disease has produced an adhesion between the retina and the vitreous. Such adhesions often persist throughout life without doing any harm. After a minor trauma which entails jarring of the vitreous, the retina tears at the place of the adhesion. Lindner's contribution was chiefly a study of the mechanics of these traumas. He states: "Only movements of the eye around its center of rotation are dangerous." The inertia of the detached and freely movable vitreous constitutes the pulling factor. In exceptional cases tears are caused by small portions of vitreous which are adherent to the retina while the bulk of the vitreous has become liquefied.

The paper contains chiefly histologic evidence in favor of Gonin's theory. The pathologic diagnosis of detachment of the vitreous is sometimes difficult. The presence of a subvitreal space filled with coagulated fluid is a reliable sign. If the subvitreal space is empty, pathologic changes of the detached hyaloid membrane or precipitates on the inner surface of the retina (thickening, breaks) indicate the presence of vitreous detachment in vivo. Using these criteria, Lindner diagnosed pathologically vitreous detachment in a large number of eyes with retinal detachment. Gonin's theory postulates that an eye with retinal detachment shows the following pathologic picture: (1) the vitreous is detached; (2) the detached vitreous is connected with the peripheral (anterior) edge of the tear, and (3) the posterior edge of the tear is free, and the subvitreal space communicates through the tear with the subretinal space. In a large number of eyes Lindner has found that these requirements were fulfilled. This is the most common mechanism of the formation of a tear, the entire mass of the still solid vitreous pulling on a relatively small retinal area. Other possible factors in the formation of a tear are the pulling effect of a small portion of vitreous adherent to the retina but unattached to the rest of the vitreous (which in these cases is liquefied) and a degenerative process in the retina without any pulling action of the vitreous. For the study of the relations between the vitreous and the retina Lindner recommends fixation after the method of Szent Györgyi (*Ztschr. f. Mikr.*, vol. 31, p. 23).

P. C. KRONFELD.

OPHTHALMOSCOPIC DIAGNOSIS OF CHOKED DISK. F. SCHIECK, Arch. f. Ophth. **137:** 203 (June) 1937.

In early stages of choked disk Schieck has observed delicate exudates within the vascular funnel which during the progression of the disease spread forward and onto the surface of the disk. There they cause bulging of the internal limiting membrane into the vitreous. During the

subsidence of the choking the limiting membrane returns relatively late to its normal position. Exudates of this form do not occur in optic neuritis or in pseudoneuritis. Gullstrand's reflex-free ophthalmoscope is less suitable for the detection of these fine exudates than is the ordinary hand ophthalmoscope. The best method of examination, of course, is that of focal illumination (ophthalmoscopy in focal light, slit lamp microscopy of the fundus).

P. C. KRONFELD.

HOMOLOGOUS CHANGES OF THE RETINAL PIGMENT EPITHELIUM AND OF THE EPITHELIUM OF THE RENAL TUBULES IN VARIOUS FORMS OF POISONING. Y. KOYANAGI and C. KINUKAWA, Arch. f. Ophth **137:** 261 (June) 1937.

The inorganic iodine preparation septojod has been known to produce, on intravenous injection, acute degeneration of the retinal pigment epithelium in man and the rabbit. Koyanagi and Kinukawa have found that septojod also affects the kidney of the rabbit, causing albuminuria, oliguria and the appearance of epithelial casts and leukocytes in the urine. Pathologically, the distal portions of the tubules are principally affected. The epithelium of the tubules contains hyaline droplets, and the cells undergo various other degenerative changes and form hyaline cylinders in the lumens, a picture which fits into that of nephrosis. Similar changes in the kidney are produced by naphthaline. The renal poisons mercury bichloride, uranium salts and guaiacol, on the other hand, cause, on injection into the carotid artery, alterations of the retinal pigment epithelium, which the authors interpret as defensive reactions, with subsequent degeneration. In all these forms of poisoning the authors consider the retinal changes and the renal changes to be homologous. Koyanagi reports the clinical and pathologic observations in one case of eclampsia. The renal observations he interprets as being typical of tubular nephrosis, while the retina only showed "secretory activity" of the pigment epithelium. "The small arteries of the choroid showed in places pronounced hyalin, the lumen obliterating degeneration." The outer retinal layers (except the pigment epithelium) were normal, a fact which, according to the authors, makes the explanation of the lesion of the pigment epithelium on a vascular basis difficult. Koyanagi and Kinukawa consider these lesions to be of toxic origin. Contrary to the reports of other authors, they found that in cases of typical albuminuric retinitis the pigment epithelium exhibits changes which are analogous to those produced in the rabbit by the aforementioned poisons. In the opinion of Koyanagi and Kinukawa, albuminuric retinitis and the homologous tubular changes are caused by toxins and not by angiospasm.

P. C. KRONFELD.

Tumors

PAGET'S DISEASE OF THE EYELID ASSOCIATED WITH CARCINOMA. A. HAGEDOORN, Brit. J. Ophth. **21:** 234 (May) 1937.

Paget's disease (in some features resembling Bowen's disease) of the eyelid was observed histologically in the eyelids removed from a patient with trachoma and xerosis. One year later the appearance of a

subcutaneous nodule necessitated exenteration of the orbit. On histologic examination this nodule proved to be a carcinoma simplex.

Histologic examination of the lid showed changes strongly resembling certain varieties of basal cell carcinoma.

The article is illustrated.

W. ZENTMAYER.

ORBITOPALPEBRAL RHABDOMYOSARCOMA. MONBRUN and G. OFFRET, Bull. Soc. d'opht. de Paris, February 1937, p. 78.

Muscular tumor is rare and one of the orbitopalpebral junction is an exception. The literature contains few reports of muscular tumor about the eye. In 1924 Redslob reported three cases of such a tumor. Mayer, Lopez-Henriquez and Cardenas Pupo found tumor of the lids. Mier reported a case of rhabdomyoma of the orbit. Monbrun and Offret removed a mass from the right palpebro-orbital junction in a child 9½ years old. The tumor grew so rapidly that the palpebral opening was greatly enlarged. Application of radium was of no avail in stopping the progress of the growth. A photograph of the child shows the tumor, and a drawing demonstrates the histologic picture of the removed tissue. The results of macroscopic and of microscopic examination are outlined in detail. Four types of cells were noted: (1) fusiform cells, which were greatly increased in number; (2) young muscle fibers, which were seen in great abundance and were very long; (3) a double striation of ordinary muscle fibers, and (4) abnormal cells—round cells with chromatin of various coloration. The authors conclude that the tumor had its origin in the superior oblique muscle.

L. L. MAYER.

Vitreous

EXPANSION PRESSURE OF VITREOUS DEPRIVED OF ITS SALTS. J. GOEDBLOED, Arch. f. Ophth. **137:** 127 (April) 1937.

The volume of vitreous deprived of its salts by immersion in distilled water increases up to 200 per cent of the original value. During this gradual loss of salts and increase in volume a low pressure due to expansion can be shown to prevail, which decreases at approximately the same rate as the volume increases.

P. C. KRONFELD.

Sympathetic Ophthalmia

PROPHYLAXIS OF SYMPATHETIC OPHTHALMIA. B. NAKAMURA and Y. UCHIDA, Arch. f. Ophth. **137:** 233 (June) 1937.

Two hundred and thirty-four patients with conditions which fulfilled the requirements of (1) perforating injury with subsequent iridocyclitis of the injured eye, (2) injury to the ciliary body, (3) sympathetic irritation of the uninjured eye and (4) sensitiveness to tuberculin were divided into two groups. The first group received prophylactic treatment consisting of injections of tuberculin (the Japanese tuberculin designated as tuberculin AO, one injection of 1 cc. a week being given) and injections of calcium iodide. In this group no patient showed

sympathetic ophthalmia, while in the second group of patients, who received no such prophylactic treatment, nine of the one hundred and thirty-nine subjects showed sympathetic ophthalmia. It is impossible to tell from the data given whether or not the two groups were—except for the prophylactic injections—treated alike, that is, whether or not the number of enucleations which might be considered as a prophylactic measure was the same in both groups. The authors are convinced of the prophylactic effect of the tuberculin treatment.

P. C. KRONFELD.

Therapeutics

CLINICAL OBSERVATIONS ON TREATMENT OF PURULENT DISEASES OF THE EYE WITH IMMUNOGEN B AGAINST THE STAPHYLOCOCCUS. G. G. BURSUK, E. I. ZAKRJEWSKAYA, B. A. SCHULTZ and K. A. HELLE, Vestnik oftal. **10:** 522, 1937.

The action of immunogen B against the staphylococcus was observed in 661 hospital patients. Of these, 137 had corneal ulcer, 90 purulent inflammation of the uveal tract, 4 dacryocystitis, 3 phlegmon of the orbit and 7 blepharitis, and for 420 the treatment was applied as a prophylactic measure.

The immunogen was given intramuscularly every other day; the dose was from 3 to 6 cc., and up to six injections were given. A general reaction was usually present; an increase in temperature, general malaise and an increase in the leukocyte count were observed in 68 per cent. There were treated: ulcers due to the staphylococcus (fifty-eight cases), ulcers due to the pneumococcus (twenty-nine cases) and corneal ulcers of mixed origin (fifty-one cases).

The most favorable results were obtained in the cases of the ulcers due to the staphylococcus; the hypopion absorbed on the fifth or the sixth day, and epithelization of the cornea occurred on the tenth to the eleventh day, with fair improvement of vision.

Treatment with milk and diphtheria antitoxin was tried for the purpose of comparison. The average length of hospitalization of the patients treated with the immunogen equaled fourteen and a half days, while patients treated with the other foreign proteins spent about twenty-six days in the hospital.

Immunogen B proved to give favorable results in traumatic iritis, while in traumatic uveitis and endophthalmitis the inflammatory process was not influenced by the treatment.

In all the cases of severe blepharitis (seven) the treatment with immunogen B gave good results. As a prophylactic measure the immunogen was injected into all patients who had to undergo an intra-ocular operation and also into all those with perforating injury of the eye. In nearly all the cases the course was an aseptic one.

The article is extensive and contains nine tables illustrating in detail the clinical data.

O. SITCHEVSKA.

Society Transactions

GERMAN OPHTHALMOLOGICAL SOCIETY

Fifty-First Annual Meeting, Heidelberg, July 6-8, 1936

TRANSLATED BY PERCY FRIDENBERG, M.D., New York

DOCENT DR. M. BÜCKLERS, *Tübingen, Reporter*

Third Scientific Session

Tuesday, July 7, 8:30 a. m.

DR. LINDBERG, Helsinki, Finland, *Chairman*

(continued from page 684)

VII. INSULIN AND THE EYE. DR. REINHARD BRAUN, Rostock.

The reader presented data based on clinical observations of 700 diabetic patients in the metabolism department of Umber in the Berlin-Westend Hospital, especially in regard to the experience with insulin in its ophthalmic therapeutic relations. He never observed any ocular damage. Even patients who had had frequent and severe insulin reactions and whose blood sugar content accordingly fluctuated markedly showed no retinal changes of any kind. Retinitis diabetica of patients under treatment with insulin runs a course which is no more hemorrhagic than is that of patients with other conditions. It is true that treatment with insulin does not seem to help the retinal disease process. On the other hand, the reader noted in 2 cases the disappearance of diabetic opacities of the lens, and in 2 other cases similar cataractous forms remained stationary for years. Insulin is of the greatest value for diabetic patients with senile cataract; since its general introduction and use the risk in operation for extraction is no greater for these patients than for patients with normal metabolism. There did not seem to be any greater tendency to hemorrhage than in the patients not under treatment with insulin. The reader was not able to get any definite idea of the relation of insulin to the intra-ocular tension. A definite observation showed, on the other hand, that hypotony of the globe is not necessarily and invariably present in diabetic coma. The usual lowering of the intra-ocular tension in this terminal condition may possibly have to be attributed to the contrast or opposition of the insulin-producing apparatus and the chromaffin system. In a case in which drops of pilocarpine had been given by mistake to a patient who was under treatment with insulin given in large doses the reader observed hypoglycemic reactions. The diabetogenic nervous diseases of the eye have evidently become very rare. In a series of diabetic patients even careful perimetric examination failed to reveal any contraction of the visual field or any scotoma. The improved treatment of diabetic patients and the use of insulin may have something to do with this. This applies also to the diabetic oculomotor disturbances which have manifestly become much less frequent than of yore.

DISCUSSION OF PAPERS VI AND VII

DR. H. GASTEIGER, Frankfort-on-Main: I was able to study a series of cases of diabetic retinitis in which the blood pressure was normal, beyond a doubt. The cases were controlled and observed as to diet, constitutional treatment and so on by the Volhard clinic in Frankfort, and the blood pressure was invariably found to be normal. So one must agree with Dr. Mylius that increased blood pressure is not to be considered the decisive etiologic factor in diabetic retinitis. It was further advised that manometric measurements of the pressure in the central retinal artery be instituted which might perhaps add to the knowledge of the origin of the ocular lesions.

DR. BARTELS, Dortmund: On the basis of my experience with extensive clinical material, I feel that the question of insulin and a possible casual relation to ocular hemorrhages has not been definitely cleared up. In 2 women with cataract, kept under observation for years before operation, hemorrhages were never found. As preliminary treatment to extraction of cataract the patients received 20 units of insulin three times daily. The patients, who had had diabetes for years, had never been subjected to treatment with insulin. After a perfectly smooth intracapsular extraction of a cataract one of the patients suffered severe retinal hemorrhages and the other, massive hemorrhages into the vitreous, which abolished sight at once, at least for a time. In the course of the next few weeks, with an insulin-free diet, the hemorrhages absorbed, and full sight was regained. In these cases it is quite possible that insulin had something to do with the hemorrhages.

DR. GILBERT, Hamburg: I had a similar experience. After the most careful preliminary treatment with insulin there was a severe hemorrhage in the eye first operated on, only the smallest part of which cleared up. Thereupon the treatment with insulin was stopped and the extraction operation on the other eye put off. When this operation was performed, after two months, the much feared complication did not materialize. This observation fits in rather well with what Dr. Bartels has told us.

DR. CAANITZ, Kiel: In a man aged 30, in the course of treatment which rapidly made his urine free from sugar, there developed, in addition to a transitory increase of hypermetropia amounting to 2 D., an isolated total paralysis of accommodation which came on suddenly, overnight, and disappeared completely in three weeks.

DR. A. PILLAT, Graz, Austria: In a study of 600 patients with diabetes who had been repeatedly subjected to a complete ophthalmologic examination, the number of ocular complications was found to be over 60 per cent, and I am inclined to attach rather more importance to the increased intra-ocular tension as an etiologic factor in the causation of retinal lesions. It is to be noted specially that in the overwhelming majority of patients with changes in the fundus observation with the Gullstrand ophthalmoscope shows retinal edema in the region of the macula or/and along smaller and larger vessels and that this edema is often present previous to the appearance of hemorrhages or white foci. In regard to damage due to insulin, long-continued use of this drug

seems without a doubt to favor the occurrence of hemorrhages in the retina and also that of pathologic lesions in the vessels of the chorio-capillaris. Cataract in diabetic patients can be operated on, after suitable preparatory internal treatment, with the same confidence as ordinary senile cataract. Repeated tonometric examination showed, in an appreciable percentage of cases, increased intra-ocular tension without any clinical signs of glaucoma, if only the blood pressure was high.

Dr. M. Bücklers, Tübingen: Dr. Braun has expressed the opinion that insulin can arrest incipient opacities of the lens in diabetic patients. The following clinical observation would seem to speak against this: The case is that of a young girl with severe diabetes (the blood sugar content was over 300 mg. per hundred cubic centimeters) who died within a year. In spite of the continued administration of large doses of insulin, typical diabetic cataract developed, first in one eye and then in the other, with the characteristic high content of fluid. The fundus showed no changes in the nature of diabetic retinitis.

Dr. W. Meisner, Cologne: Has any of my colleagues who have observed so many diabetic patients seen the very serious forms of glaucoma, first described by Salus, in which there is a marked, diffuse, hyperemia which is uniformly distributed over the iris?

Dr. C. Mylius, Hamburg: In answer to Dr. Gasteiger, the pressure in the retinal vessels was not taken. Attacks of glaucoma like those mentioned by Dr. Meisner were not observed. In answer to Dr. Pillat: There was certainly no retinal edema in the early cases, and there was no increase in the blood pressure.

Dr. Reinhard Braun, Rostock: I agree with Dr. Mylius as to the tendency to retinal edema in diabetic eyes. I have examined many eyes with the Gullstrand ophthalmoscope and could not convince myself of the presence of any edema in diabetic retinitis. I am in the fortunate position of being able to contribute some data as to the question of the etiology of diabetic retinitis, although these point in a different direction from those already presented, on the basis of 2 clinical observations.

Two patients aged 23 and 26, respectively, presented the fundus picture of typical diabetic retinitis. Both showed changes in the blood chemistry which may have given some support to a theory of the etiology. In one case the residual nitrogen content of the blood had increased to 56.8 mg. per hundred cubic centimeters, in the other to 44 mg. Both patients had a perfectly normal blood pressure. In the 23 year old woman there was clinically also a condition of renal change which probably was to be interpreted as a chronic nephritis, as the urine showed albumin, erythrocytes and casts. In the other patient the renal finding was clinically normal. Dr. Pillat's suggestion to guard against a hypoglycemic reaction by having the patient fast for a day preceding operation seems dangerous, as it is just in hunger conditions that the tendency to hypoglycemic states, i. e., insulin shock, seems most marked. In regard to the case reported by Dr. Bücklers, the question arises as to how the patient had been treated. Under Umber's influence I have come to the point of insisting on strenuous overinsulinization in cases of diabetic cataract. Whether or not, and to what extent, this was carried out in the case reported by Dr. Bücklers I cannot decide.

VIII. Familial Angiopathy in Juvenile Retinal Hemorrhages and Hemorrhages Into the Vitreous. Dr. O. Marchesani, Munich.

In the sibs of patients with adolescent intra-ocular angiopathy (periphlebitis, hemorrhages into the vitreous, retinitis proliferans) there is not infrequently found a cumulative occurrence of vascular lesions of the most varied sort. In 5 sibs, reported on in detail, the following diseases were observed: gangrene of the extremities, cerebral paralyses, ulcus ventriculi, varicose ulcers of the legs, apoplectic attacks, rheumatism and vasoneuroses.

These are the same pathologic processes which are so often to be found in adolescent persons with intra-ocular hemorrhages and which had previously induced the reader to consider the retinal disease as a manifestation of Buerger's thrombo-angiitis obliterans. Iridocyclitis, too, and rheumatism are rather frequent in cases of retinal periphlebitis and in the kinship of the patients. This observation is significant, as Rössle has lately included thrombo-angiitis obliterans in the general category of rheumatism, and many new points of view present themselves for the interpretation, *inter alia,* of the etiology of uveitis. One of the specially characteristic signs of the familial angiopathy under consideration is the occurrence of capillary lesions which are similar to those seen in severe vascular neuroses, and which, besides, are generally associated with capillary hemorrhages. The familial occurrence of the circulatory disturbances, which are to be considered as pathologically and pathogeneticaly uniform, speaks in favor of a hereditary anlage. Examination of the patients or a study of their history shows that some have had infections of one sort or another or have been subjected to toxic influences which may well have played a rôle in determining the disease. In others it is impossible to find evidence of any such irritative pathologic factors (*Reize*). The hereditary anlage seems to play the principal part. This anlage may, of course, probably be inherited in a greater or lesser quantity and/or intensity and, in a given case, suffice in the course of life to lead to manifestation of the disease, of its own account. The result of this research represents an important contribution to the question of the etiology of thrombo-angiitis obliterans.

DISCUSSION

Dr. Engelking, Heidelberg: The family trees presented by Dr. Marchesani have not convinced me. If one wishes to prove any such causal relations it is quite unpermissible to include in the clinical picture as related to thrombo-angiitis obliterans, offhand, any and every case of rheumatism and all sorts of other diseases, such as ulcers of the lower portion of the legs or open sores on the feet, without an exact characterization of these conditions. In establishing a genealogy only those cases should be used, primarily, which allow the disease picture one is looking for to be recognized in a truly typical form. In the last few years I have taken great pains in cases of retinal periphlebitis to detect signs of Buerger's disease. Although I had the cooperation of internists and other specialists, I have not so far found a single case of this condition. Nevertheless, I would not go so far as to claim (deny ?—P. H. F.) that the clinical picture of retinal periphlebitis is also seen in cases of

Buerger's disease. It must, however, be expressly stated with emphasis that periphlebitis of the retina is also a definite manifestation of tuberculosis and, as such, is frequent. I myself have records of many cases which, in this regard, leave no room for doubt.

Dr. W. Wegner, Freiburg: The family trees presented by Dr. Marchesani appear to me to prove very little. On the contrary, I feel that Dr. Marchesani has fallen into the opposite error to that which was generally made formerly, when every condition was considered as due to tuberculosis. He tries to relate everything to Buerger's disease, the essential nature of which is still obscure and insufficiently explained. I surveyed material consisting of from 65 to 70 cases of typical retinal periphlebitis. Among these cases, on careful analysis, just 2 were found which could most probably be considered as cases of Buerger's disease. Among the others, there was 1 case of severe pulmonary disease with formation of cavities; 1 female patient died of tuberculous meningitis, and a number of other patients had serious pulmonary complications which were unquestionably tuberculous. Hence I believe that the idea of a causal relation with tuberculosis is the most natural and obvious one, although one certainly cannot consider retinal periphlebitis in all cases as tuberculous.

Dr. M. Bücklers, Tübingen: At the last meeting of Bavarian ophthalmologists I presented a clinical study of 10 patients with retinal periphlebitis who had been examined with great care for the symptoms described by Dr. Marchesani as characteristic of retinal angiopathy (Buerger). In the meantime the number of patients has increased to 16, without any unquestionable cases of Buerger's disease having been found. On the other hand, 7 of 8 patients had a positive tuberculous (Meinicke) reaction. Vasoneuroses of medium severity occur comparatively often in Würtemberg. Some of the lesions in other organs reported by Dr. Marchesani are so striking that they could not possibly have been overlooked. Dr. Marchesani may be able to tell how many of Buerger's numerous patients had periphlebitis retinae. So far I cannot help having the impression, considering the small family trees adduced, that in these cases chance correlations were mistaken for system diseases, i. e., pathologically related processes.

Dr. Fleischer, Erlangen: The etiology of retinal periphlebitis and of relapsing hemorrhages into the vitreous of adolescent persons has not as yet been made clear by any means. On the other hand, Dr. Marchesani's observations are important, and it behooves all of us to institute intensive investigation in the direction pointed out by him. Still it is more than questionable whether obliterating thrombo-angiitis (Buerger) is a simple clinical and pathologic entity, specific and sharply defined, and whether it is not, perhaps, the expression of various pathologic factors. Among the latter one must certainly reckon tuberculosis. In the case first described by me the anatomicohistologic observations were those of severe pulmonary tuberculosis, to which the patient finally succumbed. In this case, then, the assumption of tuberculosis of the eye as well, and especially of the retina, seems very probable.

Dr. W. Rohrschneider, Cologne: What is the geographic distribution of cases of thrombo-angiitis obliterans in Germany? In Cologne, too, all patients with retinal periphlebitis are systematically and carefully

examined for evidence of this underlying systemic disease. So far the results have invariably been negative. What is more, internists and neurologists agree that although they are familiar with the original works of Dr. Marchesani and his collaborators, they have not been able to determine the presence of thrombo-angiitis obliterans in the population of Cologne.

DR. CAUSE: An observation which shows in the most striking way the familial relation of this vascular disease is that in the case of a student aged 24 in whom the juvenile type of retinal hemorrhages had developed seven years previously. Five months later there occurred severe apoplexy, leading to left hemiplegia, which even today makes itself felt in a very disturbing manner. In the subsequent years retinal hemorrhages occurred frequently, but there was no recurrence of any serious cerebral lesion. The patient was examined repeatedly and thoroughly last year in the Cologne clinic, and later in that of Volhard, but investigation in every direction failed to reveal anything beyond a hemorrhagic diathesis. The patient's father died at the age of 48, of periarteritis nodosa. His was the unusual case of a rare disease, in which the diagnosis was made during life in the course of an exploratory abdominal operation. The disease had dragged on for months with severe but indefinite symptoms, when acute cholecystitis developed, requiring extirpation. Even during the operation the discoloration and pallor of the large vessels had been remarked; this appearance was so striking that they could hardly be distinguished from the common bile duct. So there was undoubtedly the inheritance, at least, of a tendency to hemorrhagic diathesis in the case of the son. As a factual correction, the case had been under continuous observation for seven years, during which time the typical lesions of retinal periphlebitis, and hemorrhages of all varieties and in all layers and regions of the retina, as well as preretinal effusions of blood, had been observed.

DR. A. PILLAT, Graz, Austria: To facilitate thorough and intelligent discussion of the question which Dr. Marchesani has so happily broached, I make the suggestion that the name thrombo-angiitis obliterans be given to the condition which he considers to be retinal angiopathy. The term retinal periphlebitis, with the addition of "tuberculosa," should be applied to that condition in which, often under one's very eyes, white striations appear along the retinal blood vessels, and in which typical nodules form, generally at the bifurcations. Certainly, in view of the clinical observations and histologic research already presented, the occurrence of true tuberculous periphlebitis retinae cannot be denied. A sharp differentiation of these two diseases, including that of nomenclature, will facilitate future discussion.

DR. C. MYLIUS, Hamburg: The etiology of periphlebitis is not identical in all cases. As to the factors principally discussed here, tuberculosis and Buerger's disease, further proof will have to be furnished. For the etiology of tuberculosis in many cases, the following observation has almost the value, and is practically the equivalent, of an experiment. The case is that of a young law student, who had been treated at Kiel since 1927 for typical unilateral periphlebitis with recurring hemorrhages. At first no consititutional disorder was found. Later on the roentgen rays showed pulmonary tuberculosis. The patient

spent half a year in a sanatorium, and the process in the lungs improved temporarily. However, fresh intra-ocular hemorrhages occurred repeatedly, almost every fortnight, it was said, toward the end of 1934. Early in 1935 the clinical picture was that of typical retinal periphlebitis with numerous hemorrhages. Reye, then in Hamburg, made a diagnosis of tuberculous tonsillitis. The tonsils, which were removed, were proved to be tuberculous by histologic examination and experimentation on animals. Since the tonsillectomy there have been no more intra-ocular hemorrhages. In June 1936 the patient had completely recovered, with full vision and the fundus picture of retinitis proliferans.

Dr. O. Marchesani, Munich: Even if the genealogical charts were not convincing on account of an apparent or claimed discrepancy in the nature of the clinical pictures adduced, in numerous cases the picture is unquestionably and unequivocally pathognomonic. In a series of these cases the disorder leads to the milder symptom complexes which are characterized predominantly by the microscopic capillary observations. Rössle's pathologico-anatomic studies give reason to believe that certain forms of rheumatism are to be included in this category. Although instances of retinal angiopathy accompanied by definitely proved systemic tuberculosis are adduced, one must appeal to the general experience, which is that this observation is extremely rare in comparison with the rather frequent occurrence of my symptom complex. In Buerger's disease, retinal angiopathy has been noted by me and by other observers repeatedly. The geographic distribution of thrombo-angiitis obliterans is probably not uniform, as the disease is more frequent in the East. If it is rarely diagnosed in certain regions, the reason is probably that the clinical picture of thrombo-angiitis obliterans, in the broader sense in which it is conceived by me, is but little known as yet. Without going so far as to say that tuberculosis has no significance in the etiology of this disease, it may be stated that tuberculosis is no more important than other unspecific infections and other pathologic factors. The vascular lesions in the interior of the eye are not typically or characteristically tuberculous; they have the same appearance whether tuberculosis or some other pathologic factor has been the determining cause. I feel myself justified in this conception by my histologic studies of the eyes. All the specimens I could possibly lay hands on were submitted again, quite lately, to a succession of expert pathologists, and all agreed that the sections were not tuberculous.

IX. Clinical Course and Pathogenesis of Detachment of the Vitreous. Dr. A. Pillat, Graz, Austria.

The occurrence of this lesion in the living human eye must be considered as proved. From the anatomic point of view, i. e., according to the location of the detachment of the vitreous, one may distinguish between (1) posterior detachment and (2) anterior detachment. The first includes all detachments of the posterior portion of the limiting layer of the vitreous from the ora serrata to its attachment at the optic papilla, including the ring-shaped tearing away from the latter. Anterior detachment is accordingly the breaking loose or recession retraction of the much less extensive portion of the limiting layer of the vitreous which lies in front of the base of the vitreous. Posterior detachment

of the vitreous may be divided into five subordinate groups, viz.: (*a*) superior detachment with the subdivisions funnel-shaped and tentlike, respectively; (*b*) posterior detachment *sensu strictiori*, i. e., that which occurs in the posterior third of the globe, with its three subdivisions, cul-de-sac-like detachment, ring-shaped breaking away from the papilla and peripapillary detachment without this break: (*c*) total posterior detachment, in which usually the greater part of the mass of the vitreous sinks downward and forward (A. Vogt); but in the living eye one rarely sees what is common, probably as an artefact, in the gross anatomic specimen, viz., the entire vitreous lying directly back of the lens; (*d*) lateral detachment, which rarely comes under observation, and (*e*) inferior detachment. Misleading pictures may appear on slit lamp examination, owing to various conditions. Thus, parts of the vitreous may undergo condensation and acquire a "membranous appearance," simulating a limiting membrane, or, on the other hand, fluidification may cause the development of large, optically empty, cystic cavities. Pathogenetically one may distinguish an inflammatory and a noninflammatory (senile-degenerative) form of this lesion. The first form includes those conditions in which an inflammatory transudate or exudate in the choroid or the retina lifts up the posterior portion of the vitreous membrane from the retina, as well as those in which inflammatory products in the vitreous itself, such as white blood cells, fibrin, blood and so on, become active by bringing about a decrease of volume due to alkalinization or acidification. The noninflammatory, degenerative form is seen in myopia, in retinitis pigmentosa, in cases of detachment of the retina, in apparently sound senile eyes and after intracapsular extraction of cataract. The pathogenesis of this form can be clearly understood only if one takes into consideration three factors, the absence of which (1 and 2) or the presence of which (3) assures the normal form of the vitreous: (1) the senile shrinking of the base of the vitreous, combined with disintegration of structure in the mass of the vitreous itself; (2) senile dehydration, and (3) loosening the normal attachment of the vitreous to the retina in old age. However, the senile changes in the chemistry, form and structure of the vitreous are not based exclusively on processes in this tissue alone but depend on conditions in the choroid and retina, some of which, at least, ophthalmologists have not learned to recognize or to interpret clinically and ophthalmoscopically. Detachment of the vitreous constitutes a hazard only when pathologic adhesions have formed between the vitreous and the retina.

X. The Various Forms of Detachment of the Vitreous as Observed with the Angulated (Gewinkelten) Microscope of Lindner. Dr. H. Rieger, Vienna, Austria.

The data for 350 cases of detachment of the vitreous which originated in various ways are reported and analyzed. Referring to numerous illustrations, some diagrammatic and others drawn in color from life, the reader discussed first the three forms of detachment of the vitreous which he distinguished, viz., superior, partial posterior, and complete posterior (see also the report of the transactions of the Ophthalmological Society in Vienna on Jan. 20, 1936, in *Ztschr. f. Augenh.* **89**: 247, 1936). He then took up in detail the structure and

composition of the opacities located in the detached posterior portion of the limiting membrane. Most of the dense opacities which were formerly characterized as vitreous opacities and assigned a place in the tissue of the vitreous itself lie in the region of the detached posterior portion of the limiting membrane. These opacities have a characteristic, individual appearance which is determined largely by the age of the patient, the basic pathogenetic factor of the detachment and the region of the fundus which corresponds to the site of the opacity. The cases of detachment of the vitreous are to be divided into those in which there are holes in the region of the opacity and those in which there are no such holes. In the latter variety one is probably dealing with a simultaneous detachment of that delicate membrane composed of glia fiber cells which Salzmann has named the central glia-meniscus and which lines the walls and covers the floor of the excavation and also, although to a lesser degree, the surface of the disk itself. In juvenile subjects the limiting membrane detaches very easily, so that detachment without the formation of holes is comparatively frequent in young patients, although a similar form is seen quite often in senile subjects. In the juvenile form there is rarely an extensive dense opacity in the detached limiting membrane, whereas the senile, as well as the inflammatory, variety is characterized by dense opacities of definite form. In these cases it is probable that thickening or densification has taken place in the prepapillary glia-cuticulum.

Variations in the degree of such changes in the tissue account for the multiplicity of pictures of the opacities, increased as it is by the fact that these opacities, especially in old age, often show the formation of holes. Opacities located in the limiting membrane can also probably develop as a result of thickening of the hyaloid membrane caused by pathologic processes—mostly inflammatory—in the neighborhood of the disk, in the macular region or in the periphery of the fundus. These opacities are generally characterized by a multiplicity of holes. In conclusion, the reader discussed the significance of the condition of the detached vitreous itself for certain definite details of the clinical picture of this lesion.

DISCUSSION OF PAPERS IX AND X

DR. W. COMBERG, Rostock: My illustration of a detachment of the vitreous which showed itself in the region immediately behind the lens was published in 1924 (*Klin. Monatsbl. f. Augenh.* **72:** 695, 1924). The topographic relations could be seen and interpreted exactly with the slit lamp. The vitreous was thickened. The posterior surface of the lens lay quite free in its upper portion, bordering on a space filled with fluid and bounded posteriorly and below by the vitreous. I have maintained the conviction for some time that there is a physiologic prolapse *(Durchhängen)* of the vitreous, either of its structural framework alone, including the remains of the hyaloid artery, or, what is more probable, of the colloidal vitreous besides. Observers who use the slit lamp know that the remnants of the hyaloid artery run backward and downward, obliquely, from its point of attachment to the posterior surface of the lens. All other "contours" which are visible in the vitreous behind the lens follow the same oblique course. This uniformity of course and direction can be explained only by assuming that the regions

in question have gradually altered their position, sinking down in response to gravity in the course of life, as a result of man's upright position, and that the vitreous too sags downward (*durchhängt*). On the basis of these observations it is extremely interesting to note that most detachments of the vitreous take place above.

In view of this physiologic sag of the vitreous there must naturally result, in the course of life, a slight pull *e vacuo* which may lead to a detachment of the vitreous.

Dr. K. Lindner, Vienna, Austria: I have observed a number of cases in which the vitreous was detached to within a point close behind the lens. To Dr. Pillat's categories of degenerative and inflammatory detachment of the vitreous must be added that group in which the detachment is caused by fistulization of the globe. In cases in which this process or condition lasts for any considerable length of time the vitreous contracts, and this contraction is not fully compensated when the eye is closed, so that a detachment of the vitreous remains. Furthermore, it is not only the tongue-shaped or island-shaped attachments of the vitreous to the retina which cause retinal tears, but much more often those posterior detachments of the vitreous which show at the margin a protrusion of their edges toward the rear. Tongue-shaped islands of adhesion in cases of detachment of the vitreous cause a tearing out, with a cover to the hole *(Lochdeckel)*, which can then be found adherent to the detached vitreous, as already described with accuracy by Gonin. In case of an irregular margin at the detachment of the vitreous so-called flap tears *(Lappenrisse)* develop, the flap remaining attached to the detached portion of the vitreous. In almost every case of retinal detachment one finds a detachment of the vitreous as one of the preceding, partial, predisposing pathogenic factors. The retinal lesions, for the greater part degenerative, must then occur as the exciting factor at the point on which the vitreous exerts traction, either as an island or in the form of an irregular protrusion. Finally, it is the elastic shock of the vitreous which causes, in the first instance, a hole-shaped tear and, in the second, a flap-shaped rip (*Einriss*) of the retina. I formerly shared the opinion that retinal detachment could take place spontaneously in the presence of fluidification of the vitreous as a result of impact and traction on degenerated portions of the retina by the residual shreds and clumps (*Ballen*) of vitreous. This is, however, not the case, or at least very rarely so, as individual masses of vitreous have little kinetic energy or force and, besides, run out toward the optic papilla to a flat base, as the shock effect of the mass of vitreous in motion spreads and becomes distributed. A primary retinal tear can ensue only when this shock action is concentrated on a sharply limited area, such as the island-shaped adhesion which in cases of detachment of the vitreous persists behind the general margin of the detachment, or, again, on a marginal zone of adhesion which runs out toward the disk. It was not by any means theoretical considerations, but rather a careful and thorough sampling and analysis of all the specimens having a bearing on this point which brought these facts to my special attention and induced me, in view of the generally meager results of the interpretation of histologic observations in changes in the vitreous, to look for a method of detecting the presence of a detachment of the vitreous in the living subject as well. There is such a method today.

On the basis of this ophthalmologists now know that the great majority of spontaneous retinal detachments take place only in eyes which have already shown a detachment of the vitreous, which corroborates part of Leber's original theory. However, we are not yet able to recognize those cases in which the eye is especially jeopardized owing to the presence of a detachment of the vitreous, as the apparatus for examination now at our disposal does not enable us to inspect completely the boundaries of a detachment of the vitreous.

DR. K. VELHAGEN, Halle-on-Saale: I have been interested for some time in the senile retinal atrophy mentioned by Dr. Pillat, a condition which has been described histologically more often and more thoroughly than ophthalmoscopically. According to my observations, this lesion lies predominantly, not extremely peripherally but in a somewhat intermediate position, in a ring. This is probably the limit of the region supplied by the arteria centralis retinae. Farther toward the periphery the blood supply comes from the ciliary circulation. The nature of the intermediate region, which is particularly exposed to atrophy, probably explains the relative infrequency of tears at the ora serrata and the frequency of tears in the intermediate zone. The observation of detachment of the vitreous after removal of the lens in its capsule reminds me that mention has been made repeatedly, in the Spanish literature, that retinal detachment too is decidedly more frequent after intracapsular than after extracapsular extraction of cataract.

DR. A. PILLAT, Graz, Austria: I feel that the condition illustrated by the sketch shown by Dr. Comberg might correspond to an anterior detachment of the vitreous, a condition which I have never seen in my clinical material. I agree with Dr. Velhagen that the senile changes in the retina are located principally in the region of the equator or at least are most marked in that area of the fundus. A very convincing and clear picture of senile pigmentary degeneration is presented.

DR. K. RIEGER, Vienna Austria: In closing, in regard to the way in which detachment of the vitreous comes about, one must distinguish sharply between a spontaneous detachment and one that takes place in a globe which has been opened previously. As to the etiology of spontaneous detachment, it is worth while to refer to the papers presented in January and March 1936, at the meeting of the Ophthalmological Society in Vienna (*Klin. Monatsbl. f. Augenh.* **96:** 396 and 695, 1936) and to an exhaustive work on the subject which is to appear, shortly. The detection of a detachment of the vitreous after extraction of cataract has been carried out does not permit one to infer anything about an eventual causal relationship with the operation. The detachment may have been present as a myopic or a senile complication before the surgical procedure, without any possibility of ascertaining its presence. My own observations carried out in the examination of that eye of patients with a cataract which still permitted a view of the fundus, speak uniformly in favor of this view. Furthermore, comparative investigations of eyes after intra-capsular and extra-capsular extraction of cataract, respectively, are hardly possible, as the view of the fundus in the latter category is often insufficient for a thorough investigation of the vitreous.

XI. Measurement of Illumination of Optotype Charts. Dr. T. Graff, Rathenow.

Notation of the illumination of test charts is quite insufficient to indicate their luminosity. In the case of charts reflecting the illumination one should state the luminosity per square centimeters, at right angles to the chart, i. e., the density of illumination, in Stilb units, which is, besides, the natural way and the easiest for measurement in the case of translucent test charts. To determine the density of illumination by means of the formula illumination density in apostilb units = illumination intensity in lux units in which 1 stilb = $10^4\pi$ apostilbs is too uncertain, as the quality and quantity of light reflected from the chart would have to be considered more carefully. With an illumination density of around 0.05 stilbs and a contrast of the dark figures against the bright background amounting to 99.75 per cent, the influence of fluctuations in illumination and, depending on these, of changes in the diameter of the pupil, is minimal.

XII. Clinical Experience with the New Zeiss Projection Perimeter (According to Maggiore). Dr. H. Serr, Jena.

The reader has carried out many observations on normal subjects and on patients with defects in the central and peripheral field of vision. These observations were checked by simultaneous study with the automatically registering Loew perimeter, made by Jung in Heidelberg, and/or with the Bjerrum screen. Under the experimental conditions chosen, the following results were noted: 1. The determination of the peripheral limits of the visual field with the projection perimeter is effected with surprising ease, so that in the case of children, nervous patients and so on one can frequently get definite and reliable statements, whereas this is not feasible with the Loew perimeter. The patients also tired less easily. The peripheral fields for white showed, with the projection perimeter, perceptibly greater extension in the temporal half and below, in normal subjects, than with the Loew perimeter with bright daylight. The projection perimenter meets all the clinical requirements of color perimetry of the peripheral field, but the usual clinical requirements need overhauling from the scientific standpoint. The projection perimeter enables one to determine the peripheral limits of the field with lowered illumination of the test objects, without circumstantial preparatory arrangements, and this may in many cases give data of clinical importance. The instrument is particularly well suited to detect and map out small scotomas in the central visual field and so can well take the place of the Bjerrum screen.

XIII. Can Central Scotomas be Recognized Objectively? Dr. Heinrich Harms, Berlin.

The investigation started from the consideration that defects in the central visual field, if caused by lesions situated between the retina and the geniculate body, would have to go hand in hand, clinically, with a loss of central pupillomotor sensitivity, or at least its diminution, on account of the intimate connection between the visual pathways and the ascending light reflex path. The experimental principle of alter-

nating and varying illumination used by Hess in studying hemianopic pupillary stasis was adapted to the research in question by the construction of suitable opaque diaphragms or shutters (*Blenden*). These permit a change from central to peripheral illumination of the retina and allow one to compare the pupillary reaction which takes place in each case. The extent of this pupillary reaction can be equalized by making the central stimulation field smaller or by lowering the intensity of its illumination. The quotient of the light volumes used centrally and peripherally characterizes the pupillomotor conditions of the eye under examination. Preliminary studies showed that, with a marked degree of constancy in a given eye, the quotient of subjects with normal vision varies greatly in individual cases, viz., from 0.6 to 0.07. Differences between the two eyes of the same person up to one third of the higher value were found. In testing macular degeneration the large majority of subjects were found to have the difference in quotient which was to be expected from the amount of vision present. In some the quotient was found to be greater in the better-seeing eye. This could be explained by differences in the diffusion of light in the refracting media or possibly by separated lesions of the light path and the light reflex path, respectively.

XIV. The Question of Accommodation. Dr. H. Erggelet, Göttingen.

Although the axial length of the eyeball is of basic significance in the question of the origin of accommodation, frequency curves and graphs of ocular length have been lacking up to the present. The curve constructed by E. Tron is based on values for length which have been calculated from the determining sections of the succeeding optical planes in connection with the previously determined state of accommodation of the eye. The reader, together with Geserick, took direct measurements of the anteroposterior diameter of 122 eyes and plotted a curve of frequency. In contrast to the research material of an ophthalmic clinic, a deleterious selection was avoided by using for measurement only the cadaveric eyes from a pathologic institute. Hence one may expect a statistical result which holds good for the population in general. With a group division of 0.5 mm. the curve came out practically symmetrical, but perhaps with somewhat high peaks. The frequency polygon (*Vieleck*) varied very slightly from the binomial curve route. The variation area extended from 21.7 to 28.75 mm. The average axial length amounts to 24.68 ± 0.089 mm. The spread (σ) $= \pm 0.988$. These values were then compared with the figures represented by the curves of Tron.

DISCUSSIONS OF PAPERS XI TO XIV

Dr. H. Lauber, Warsaw, Poland: In order to obtain a uniform illumination of the perimeter arc and to be able to investigate always under the same conditions, the fields should be taken in the dark room, a broad perimeter arc which is illuminated just enough to preserve the light adaptation of the eye being used. By means of a punctal light projection which can be used for determining the peripheral limits of the field, as well as for campimetry with the Bjerrum screen or with

Lloyd's campimeter, the change in position of the test object takes place noiselessly and cannot be in any way checked up by the subject who is being examined. Unfortunately the apparatus has been almost completely relegated to oblivion [sic].

DR. LÖHLEIN, Berlin: Can this method of detecting the presence of a central scotoma by quantitative comparative determination of pupillary reaction be applied also to intelligent children of from 5 to 8 years of age? If that were the case it would throw light on a whole series of basic problems connected with amblyopia due to squint. For instance, if one found no increase in vision after an early and systematic course of fusion training therapy, one could take it for granted that the amblyopia was the primary factor and the strabismus secondary. This conclusion would be essentially corroborated if it could be shown that the light reaction relations between the macula and the periphery, as well, remained uninfluenced. In the other event, that of secondary amblyopia (ex anopsia), the method might allow one to draw conclusions as to whether the site of exclusion due to nonuse is to be sought in the periphery, where optical visual and pupillomotor paths are still united, or centrally. One would suppose that both localizations are possible. Finally, it is not at all unusual to find in the course of fusion training that central vision shows no improvement as measured by the test chart. In spite of this the child who appeared quite helpless on the first day on which the bandage was applied quickly acquires a self-confident and easy bearing. These are evidently cases in which there was no increase in the function of the maculopapillary bundle, while the peripheral portions of the retina gained materially by practice. If anything of this sort does take place, it might perhaps be demonstrated by a corresponding reversal in the relation of the pupillary reaction produced by stimulation of the macular portion of the retina to that produced by stimulation of the peripheral portion. There seems to be a wide field for the application of this new method.

DR. BEST, Dresden: The Zeiss projection perimeter, which I have been using for two months, is unexcelled for clinical purposes. Without going into details, there is one point on which one cannot agree with Serr. It is true that one can map out the most minute scotomas with the projection perimeter, but in this respect the Bjerrum screen is still superior, if the test object is projected on it. This is partially due to the fact that with the screen one can use distances of from 1 to 5 meters and correspondingly smaller test images.

DR. ENGELKING, Heidelberg: The perimeter used by Dr. Serr is undoubtedly useful for many purposes, but I cannot agree with Dr. Best that the instrument cannot be improved on. For one thing, note that the illumination of the perimeter arc is very uneven, as a result of which the white test objects appear relatively much brighter when centrally located than at the periphery. Even cursory inspection shows that the red and/or green markers appear slightly yellow in the periphery and are, accordingly, not invariable. Furthermore, they have not the same luminosity and, again, differ in luminosity from the blue objects. So in studies of the field with these color objects and this perimeter one is testing not the color sense alone, but at the same time contrasts in luminosity, besides which the luminosity of the color objects is not the

same as that of the perimeter arc. These are all requirements which must be met by an up-to-date perimeter. Finally, the physiologic saturation of the two colors of a pair, e. g., the red-green objects, is not the same, so that the limit for green appears more restricted than that for red, which should not occur if persons have a color sense in which green and red are coupled.

DR. OHM, Bottrop: It is not generally known that opticokinetic nystagmus can be used very well for the objective detection of central scotomas. If the scotoma is large, the revolving drum is screened off more and more from the periphery by means of four screens to a point at which the nystagmus disappears. The course of severe retrobulbar neuritis can in this way be represented in curves of the opticokinetic nystagmus. As a region of 3 degrees is required for the elicitation of this reaction, one has to use another procedure for small scotomas. If, by means of a mirror, one throws a bright image into the eye in such a way that it appears to be in line with the middle of the rotating drum, the opticokinetic nystagmus disappears immediately the moment the light flashes, if it attracts attention sharply. My smallest marker is 0.25 mm. and corresponds to a visual angle of 1.75 minutes of arc. If that does not suffice, larger markers are used until one reaches the point at which opticokinetic nystagmus is inhibited. In this way one can get an objective conception of the visual acuity even in cases in which other methods fail completely, as in the case of a patient with only one eye and a very small pupil with adhesions. The amblyopia of a squinting eye can also be represented by means of opticokinetic nystagmus. In order to make full use of my method, nystagmography, in addition, is requisite.

DR. PAUL, Lüneburg: Anatomic determination of the axial length of the eyeball by measurement of the eyes of cadavers is of value. The eyes can, however, be fully used only if the total refraction of the anatomically measured eye, as well as that of the cornea, has been previously determined. For the axial length varies greatly not only with varying refraction of the globe but with variation in the composite structure of the optical system and identical refraction.

DR. H. SERR, Jena: In my comparative studies of central scotomas I have used, in addition to the projection perimeter, the usual technic of the Bjerrum screen with ordinary, i. e., not projected, test objects. Furthermore, I purposely abstained from using any special artificial lighting arrangements, because, for the time being I wished to test the new apparatus only under such conditions as could be easily provided by the practicing physician. In regard to color perimetry, I have already noted sufficiently in my paper that the projection perimeter in its present form meets only the clinical requirements which have been usual up to the present time and not those which must be considered from the scientific standpoint. Dr. Engelking's valid adverse criticism of the apparatus was to be expected, a priori.

DR. H. HARMS, Berlin: The stimulus for the development of my method was given by occupation with the scotomas of subjects with strabismus. It was intended primarily to determine in this way the location of the point where the strabismic image was suppressed. For

reasons previously stated, opticokinetic nystagmus is not suited to this purpose. The examination of children by my method depends for its success first on whether they have sufficient attention for reliable fixation and then on the development of a method which will insure reliable fixation by the amblyopic eye during the test. A suitable procedure is being worked out. The evident improvement in visual ability of amblyopic children after exclusion of the better eye under a bandage, without any corresponding increase in visual acuity, may perhaps depend on diminution in size of an existing scotoma.

Dr. H. Erggelet, Göttingen: I feel that the output of our measurements would be enriched if we had definite basic and standard values of accommodation. Hence, as was said, I have desisted from building on unreliable figures. As to the value of the graph presented, in studying the problem of refraction no valid conclusions can be reached without exact data on the axial length of the eyeball. In a roundabout way, far-reaching deductions have been made by replacing unknown values by accepted ones in the reckoning. The results have no solid basis. The curves for the axial length of the eyeball, e. g., that of Tron, may be, but are not necessarily, accurate. Research workers should welcome all the more the curves presented, which have a definite basis. They would be unusually valuable for comparison and check-up and would in a given case support the entire study if both curves agreed.

(*To be Continued*)

Book Reviews

Internal Diseases of the Eye and Atlas of Ophthalmoscopy. By Manuel Uribe Troncoso, M.D., formerly Professor of Ophthalmology, New York Post-Graduate Medical School and Hospital; Instructor in Ophthalmology, College of Physicians and Surgeons, Columbia University; Assistant Ophthalmologist, Presbyterian Hospital, New York. Price, $15. Pp. 530, with 239 engravings, including 95 figures on 82 full-page color plates. Philadelphia: F. A. Davis Company, 1937.

This stately volume is not only a valuable addition to the series of treatises on the examination of the fundus oculi and the accompanying atlases with which the names of Liebreich, Oeller, Haab, Adam, Oatman and many others are connected. It registers, in this reviewer's opinion, a distinct advance in method of presentation, in scope and inclusiveness and in correlation of the ophthalmoscopic findings with the lesions, the clinical history and the symptomatology. With a very practical, as well as a logical, mind, Professor Troncoso has, by his systematic classifications, put one in a position to make a positive diagnosis or at least, as he states, a diagnosis by exclusion. In this respect the chapter on the normal retinal circulation and its disturbances and their relations to local and systemic disease is worthy of special mention as one of the greatest value not only for the student but for the more advanced and experienced ophthalmologist as well. The optical principles involved in the examination of the transparent media and the intra-ocular tissues are explained clearly, succintly and without recourse to higher mathematics. The technic in these procedures and in others, such as the determination of differences in level, is illustrated by simple diagrams. The chapter on the ophthalmoscope deals, as is usual, with the history of the instrument, but this subject is brought up to date in a welcome description of the latest types of this invaluable aid to ophthalmic diagnosis. The various electrically illuminated models, the binocular ophthalmoscope and ophthalmoscopy with red-free light are considered. There is a valuable section on the physiology and pathology of the field of vision and the various methods of examination in health and disease, including stereoscopic campimetry, angioscotometry and the technic of perimetry. The use of the termination "osis" for hereditary, degenerative, or/and senile changes in contradistinction to inflammatory processes undoubtedly conduces to the logic of teaching and to simplifying classification. Although, as the author modestly states, this terminology has not yet been generally accepted, some ophthalmologists, notably, Mawas, have adopted and made good use of it. The colored plates are well chosen and of uniform excellence in color, drawing and reproduction. In many cases the views of the fundus are supplemented by pictures of microscopic sections of the normal and/or diseased part concerned. Most of these pictures of histologic sections are printed in a position the reverse of that usually shown in textbooks, e. g., with the excavation of the optic disk pointing toward the bottom of the page, the trunk of the nerve pointing upward, and so on. As the lettering is, as usual, upright, this is confusing until one becomes accustomed to it. A special chapter is devoted to an instructive consideration

of pituitary tumors and their sequelae in the form of pressure atrophy, a good survey of this topic being given. In the section on retinal detachment, the material of which is well up to date, an outline is given of the modern operations. Injuries of the choroid, retina and optic nerve so far as they relate to ophthalmoscopy are discussed and described, and there is some comment on the mechanism, etiology and general pathologic features. In a subsequent edition, which may well be called for, one might suggest additional pictures of the fundus illustrative of later stages of retinal arterial obstruction, especially embolism, and of circinate retinitis, Coat's massive exudation, angioid streaks and the condition of the disk in wood alcohol amblyopia.

PERCY FRIDENBERG.

Short-Wave Diathermy. By Tibor De Cholnoky, M.D., Associate in Surgery, New York Post-Graduate Medical School and Hospital. Price, $4. Pp. 310, with illustrations. New York, Columbia University Press, 1937.

The subject is treated under the following headings: A historical outline, the physical aspects of short wave diathermy, experiments in bacteriology and on animals, the technic of short wave diathermy and the clinical application.

De Cholnoky emphasizes that it is wrong to say that short wave diathermy is simple and that it carries no dangers; it is not magic but a new agent derived from a form of electric energy. It is not a cure-all and should not be applied unless its physical action is understood and the pathologic condition of the patient to be treated is clear. Heat is the most important result, and hence this treatment is indicated in inflammatory disorders. It is superior to other forms of heat therapy because of its deep action. The specific action on bacteria has not been proved. While diathermy gives more heat on the surface, short wave diathermy allows the penetration of heat and a uniform action in the deeper structures, provided the application of the electrodes is correct. The proper application of the electrodes is most important to prevent the current from concentrating. The subject of short wave diathermy is not well understood, and the contraindications are not yet clearly defined. The great stumbling block has been the inability to measure the degree of heat obtained, and the result can be estimated only by the patient's subjective sensation to heat.

The author finds that short wave diathermy is to be recommended in the treatment of common colds and in sinus disease. For such conditions a special electrode is best.

This is a clearly written description of short wave diathermy and can be recommended to those who are interested in this form of treatment.

Unfortunately, the diseases of the eye are but briefly mentioned. The author apparently has had little experience with the action of this form of therapy on the eye, though the treatment has been carried on in a number of continental clinics. The great difficulty, again, is the inability to judge the degree of heat developed in the interior of the eye, and the possible deleterious effect on the lens.

The chapters on the clinical application will undoubtedly be enlarged and the indications and contraindications defined when more experience has been obtained.

ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

Honorary President: Prof. F. de Lapersonne, 217 Fauborg St. Honoré, Paris.
President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the President, Dr. P. Bailliart.

International Ophthalmologic Congress

Secretary: Dr. E. Marx, Costzeedijk 316 Rotterdam, Netherlands.
Place: Cairo. Time: Dec. 8-14, 1937.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. P. E. H. Adams, 6 Holywell, Oxford.
Secretary: Dr. Thomasina Belt, 13 Mitchell Ave., Jesmond, Newcastle-on-Tyne.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. Lohlein, Jena.
Secretary: Prof. A. Wagenmann, Heidelberg.

Midland Ophthalmological Society

President: Dr. T. E. Ashdown Carr, 34 Charnwood St., Derby, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.
Time: Oct. 1, 1937.

Ophthalmological Society of Egypt

President: Prof. Dr. Sayed Abdel Hamid Soliman Pasha, Faculty of Medicine, Cairo.
Secretary: Dr. Abdel Fattah El Tobgy, 3 Midan Soliman Pasha, Cairo.
Time: March 1938.

Ophthalmological Society of the United Kingdom

President: Mr. Gordon M. Holmes, 9 Wimpole St., London, W. 1.
Secretary: Mr. J. D. M. Cardell, 27 Weymouth St., London, W. 1.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Oxford Ophthalmological Congress

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Time: July 8-10, 1937.

Polish Ophthalmological Society

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warszawa.
Place: Lindley'a 4, Warszawa.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. Ransom Pickard, 31 East Southernhay, Exeter, England.
Secretary: Dr. A. Rugg-Gunn, 35 Harley St., London, W. 1.

Société Française d'Ophtalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris, 7è.

Society of Swedish Ophthalmologists

President: Prof. K. G. Ploman, Stockholm, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

Tsinan Ophthalmological Society

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
Place: San Francisco. Time: June 13-17, 1938.

American Academy of Ophthalmology and Otolaryngology, Section on Ophthalmology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

American Ophthalmological Society

President: Dr. Frederick H. Verhoeff, 243 Charles St., Boston.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Place: San Francisco. Time: June 9-11, 1938.

National Society for the Prevention of Blindness

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.
Place: New York. Time: Oct. 6-8, 1937.

SECTIONAL

Central Wisconsin Society of Ophthalmology and Otolaryngology

President: Dr. E. S. Schmidt, 107 E. Walnut St., Green Bay.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Rd., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. William Donoher, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.
Place: San Francisco. Time: June 22-25, 1938.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. W. Howe, 740 St. Helens Ave., Tacoma, Wash.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Frank W. Broderick, 501 Central Trust Bldg., Sterling, Ill.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501—7th St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Secretary-Treasurer: Dr. Andre Cortopassi, 703 Second National Bank Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. J. H. Judd, 107 S. 17th St., Omaha, Neb.
Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.
Place: New Orleans. Time: Nov. 30-Dec. 1-3, 1937.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. John Hunter McRae, 26 Sheldon Ave., S. E., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. Wearne Beals, Weber Bldg., DuBois.
Secretary-Treasurer: Dr. C. Wearne Beals, Weber Bldg., DuBois.
Place: Indiana, Pa. Time: Oct. 21, 1937.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Charles T. Flynn, 41 Trumbull St., New Haven.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Place: New London. Time: February 1938.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. John King, Thomasville, Ga.
Secretary-Treasurer: Dr. Mason Baird, 384 Peachtree St., Atlanta, Ga.
Place: Augusta. Time: May 1938.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. E. E. Holland, 51 S. 8th St., Richmond.
Secretary: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: April 6, 1938.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. James A. Downing, 406—6th Ave., Des Moines.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
Place: Des Moines. Time: Nov. 18, 1937.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. C. A. McWilliams, G. and S. I. R. R. Bldg., Gulfport, Miss.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Place: New Orleans. Time: Spring, 1938.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. F. N. Smith, Grand Rapids Clinic, Grand Rapids.
Secretary: Dr. Dewey R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Walter E. Camp, 1918 Medical Arts Bldg., Minneapolis.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Edward S. Murphy, Northern Pacific Hospital, Missoula.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Oram Kline, 414 Cooper St., Camden.
Secretary: Dr. James S. Shipman, 542 Cooper St., Camden.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Marvin F. Jones, 121 E. 60th St., New York.
Secretary: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Time: May 10, 1938.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. J. M. Lilly, 302 Old St., Fayetteville.
Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.
Place: Charlotte. Time: October.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. Rosenberger, 221—5th St., Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, 514—6th St., Valley City.
Place: Bismarck. Time: May 1938.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George Truluck, Orangeburg.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.
Place: Columbia. Time: Nov. 9, 1937.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Kate Savage Zerfoss, 165—8th Ave. N., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 805 Medical Arts Bldg., Memphis.
Place: Nashville. Time: April 12-13, 1938.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.
Place: Fort Worth. Time: Dec. 11 and 12, 1937.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. M. H. Hood, 505 Washington St., Portsmouth.
Secretary-Treasurer: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. James A. Fisher, 501 Grand Ave., Asbury Park, N. J.
Secretary: Dr. William F. McKim, 488 Sanford Ave., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Andersen, 106 S. Main St., Akron.
Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. William C. Warren Jr., 478 Peachtree St., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Henry F. Graff, 513 N. Charles St., Baltimore.
Secretary: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Ivan J. Koenig, 40 North St., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Thomas D. Allen, 122 S. Michigan Blvd., Chicago.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Webb Chamberlin, 7405 Detroit Ave., Cleveland.
Secretary: Dr. Walter H. Rieger, Hanna Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. Paul Moore, Republic Bldg., Cleveland.
Secretary: Dr. G. Leslie Miller, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. C. E. Silbernagle, 247 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. D. M. Johnson, 327 E. State St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. Edgar G. Mathis, 416 Chaparal St., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 416 Chaparral St., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Lester H. Quinn, 4105 Live Oak, Dallas, Texas.
Secretary: Dr. J. Dudley Singleton, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. William Fowler, 1424 Maccabee Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. J. D. Carroll, 102—3rd St., Troy, N. Y.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 306 W. Broadway, Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. L. Allen, 1215 Walker Ave., Houston, Texas
Secretary: Dr. George C. Farrish, 1625 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. C. P. Clark, 24 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. O. S. Gilliland, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. F. C. Hertzog, 117 E. 8th St., Long Beach, Calif.
Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. Isaac H. Jones, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Earle Breeding, 1801 I St., N. W., Washington.
Secretary: Dr. Elmer Shepherd, 1606–20th St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Thomas A. Judge, 735 N. Water St., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 208 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Minneapolis Ophthalmological Society

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

Montgomery County Medical Society

Chairman: Dr. A. G. Farmer, 1040 Fidelity Bldg., Dayton, O.
Secretary-Treasurer: Dr. Rome M. Webster, 663 Reibold Bldg., Dayton, O.
Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. Stuart Ramsay, 1496 Mountain St., Montreal, Canada.
Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Guy Maness, Nashville, Tenn.
Secretary-Treasurer: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Society for Clinical Ophthalmology

Chairman: Dr. James W. White, 15 Park Ave., New York.
Secretary: Dr. Rudolf Aebli, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

new York Society for Clinical Ophthalmology

President: Dr. Mark J. Schoenberg, 1160 Park Ave., New York.
Secretary: Dr. Jesse Stark, 45 Park Ave., New York.
Place: Squibb Hall, 745 5th Ave. Time: 7 p. m., first Monday of each month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. A. J. Young, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Ophthalmological Society of the University of Pittsburgh

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, 351—5th Ave., Pittsburgh.
Time: Second Monday in November, January, March and May.

Passaic-Bergen Ophthalmological Club

President: Dr. John S. Van Winkle, 297 Broadway, Paterson, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, 351—5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. A. F. Bagby, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. Leslie Charles Drews, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Joseph W. Crawford, 490 Post St., San Francisco.
Secretary: Dr. Russell Fletcher, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Raymond A. Lower, 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Louis A. Parsell, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. Gordon D. Hoople, 110 Medical Arts Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St., W., Toronto, Canada.
Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. G. Victor Simpson, 1710 Rhode Island Ave., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Frank D. Costenbader, 1726 Eye St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.